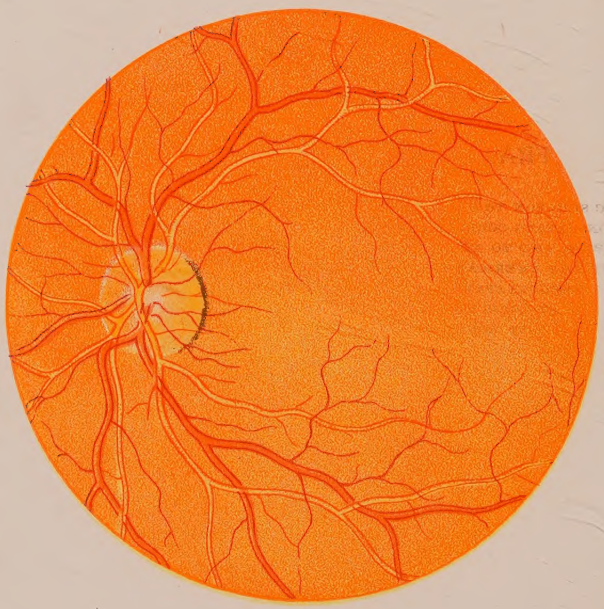
LESSONS ON THE EYE

HENDERSON.



NORMAL FUNDUS.

LESSONS ON THE EYE

FOR THE USE OF

UNDERGRADUATE STUDENTS

BY

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PREFACE.

Student's manuals on diseases of the eye are, as a rule, either exhaustive treatises in fine print or condensations of the *entire* science of ophthalmology. The authors of these works seem loath to omit any knowledge which, as specialists, they have acquired. The result is that subjects which the general practitioner never attempts to master are given as much space as those with which he should be familiar.

It should be the purpose of a medical school to provide its graduates with an equipment which will best meet the demands of general practice, and I recognize that there is enough matter to fill the course to overflowing, that is of more importance than the layers of the retina or the formula for calculating the index of refraction of a transparent medium.

The only claim to originality made for this work lies in its omissions. Minute anatomy, the fitting of glasses, skiascopy, ophthalmoscopy and kindred subjects have been left out intentionally, as I believe they belong to post-graduate instruction. I have also slighted those diseases which have to be diagnosed with the ophthalmoscope, as I doubt the diagnostic value of an ophthalmoscope in the hands of the average practitioner.

It is not my desire to minimize medical education but rather to increase the *useful* knowledge of the graduate by selecting that which will be of the most service to him, at the same time giving him as much as the undergraduate student can reasonably be expected to learn in the limited time allotted to the eye in our medical schools. The use of these printed notes enables the teacher to devote much time

to quizzing which would otherwise be spent in lecturing. They also enable the student to dispense with his inaccurate and misleading classroom notes. The subject has been divided into twenty-eight lessons, or one lesson for each week of a seven months' session. In the spelling of such words as oxid, quinin, morphin, sulfate, etc., the rules adopted in 1891, by the American Association for the Advancement of Science, have been followed.

CENTURY BUILDING, ST. LOUIS, MO.

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LESSON I.

ANATOMY OF THE EYE.

THE ORBITS.

These four-sided, pyramidal or conical cavities, a little over an inch and a half deep, are formed by seven bones: frontal, sphenoid, ethmoid, superior maxillary, palate, malar and lacrymal. On the inner wall of the orbit is the groove,

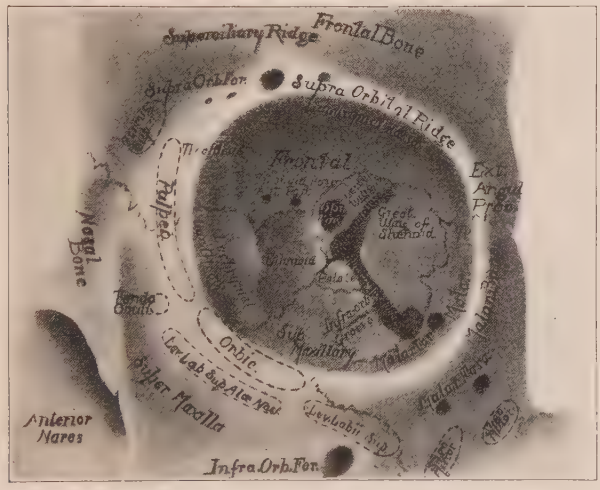

FIG. 1.—Left orbit.

formed by the lacrymal bone and the nasal spine of the superior maxillary, in which is lodged the *lacrymal sac*. In front of this groove is the insertion of the orbicularis, the muscle which closes the eyelids, and behind the groove is the insertion of the tensor tarsi or Horner's muscle which

holds the lids close to the globe. In the angle formed by the roof of the orbit and the nasal wall, and a short distance behind the orbital rim, is the loop or pulley through which passes the tendon of the superior oblique muscle. In the angle formed by the roof and the temporal wall and just under the edge of the orbit is a fossa which holds the lacrymal gland. At the junction of the inner and middle thirds of the superior orbital rim is the *supra-orbital notch* or foramen through which passes the supra-orbital nerve, artery and vein. Below the infra-orbital rim is the *infra-orbital foramen,* which is the termination of the canal of the same name. Near the apex of the orbit and between the great and lesser wing of the sphenoid bone is the *sphenoidal fissure,* which transmits the third, fourth, the ophthalmic division of the fifth and the sixth nerves and the ophthalmic vein. The apex of the orbit corresponds to the *optic foramen,* a cylindrical canal in the lesser wing of the sphenoid bone, which transmits the optic nerve and ophthalmic artery.

Extending forward and outward from near the apex is the *spheno-maxillary fissure.* It lies between the lower border of the great wing of the sphenoid bone and the maxillary bone, and transmits the infra-orbital vessels and several nerves. In the middle of the orbital floor is the *infra-orbital* groove which terminates in the *infra-orbital canal.* The bones are covered by periosteum and the orbital space not filled by the eyeball, nerves, muscles and vessels, is occupied by fat and connective tissue. This connective tissue becomes thickened in parts so as to form sheaths for the muscles and optic nerve. It also develops a membrane which spreads over the eyeball, from the entrance of the optic nerve to within three millimeters of the cornea where it becomes inseparably mingled with the conjunctiva. This membrane is called *Tenon's capsule.* It is loosely connected to the episclera, the space between them serving as a lymph chan-

nel. The *ophthalmic artery,* a branch of the internal carotid, supplies blood to the orbit and its contents.

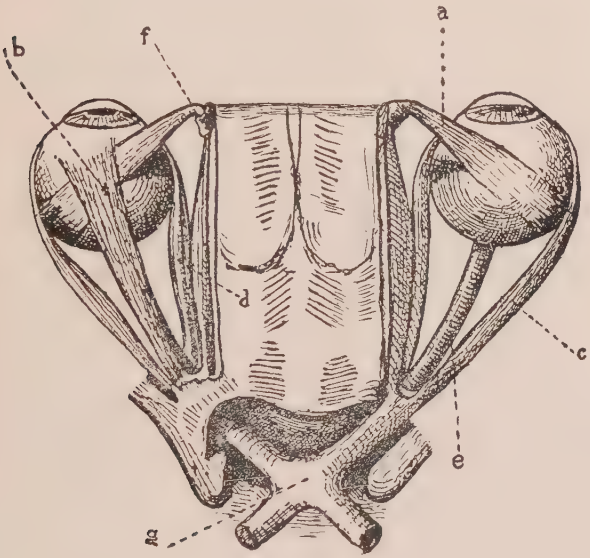

FIG. 2.—The ocular muscles seen from above. a, Superior oblique; b, Superior rectus; c, External rectus; d, Internal rectus; e, Optic nerve; f, Pulley of superior oblique; g, Optic commissure.

THE OCULAR MUSCLES.

The ocular muscles are six in number, the internal, external, superior and inferior recti, and the superior and inferior oblique. All except the inferior oblique arise from the apex of the orbit around the optic foramen. The inferior oblique arises from the floor of the orbit, in a slight depression in the superior maxillary bone, near the lacrymal groove. All the ocular muscles, after piercing the capsule of Tenon are inserted in the sclera, the four recti at points varying from 5.5 to 7.5 millimeters from the cornea.

Though the superior oblique arises at the apex of the orbit, the direction of its force is changed by passing through the pulley, before mentioned, which is situated in the angle formed by the roof and the nasal wall of the orbit. From this pulley its direction is backward and outward and passing under the superior rectus, it is inserted into the outer side of the globe, more than half of the tendon being inserted

Fig. 3.—Muscles of the left eye.

back of the equator. The inferior oblique runs backward and outward, and passing between the orbital floor and the inferior rectus, it is inserted into the outer side of the globe, more than half of the tendon being back of the equator at a point below the superior oblique. In addition to its scleral attachment, some fibers from the superior rectus and its sheath pass to the conjunctival fornix and to the top of the tarsus, by which means these structures are moved in harmony with the upward movement of the eyeball. The ter-

mination of the inferior rectus is similar to that of the superior. In the same way fibrous bands are given off from the sheaths of the internal and external recti and pass laterally to the bones and soft parts of each side of the orbit. The *levator palpebrae superioris* muscle, which lifts the upper lid, owing to its location, is best described with the ocular muscles. It arises at the apex of the orbit and passes for-

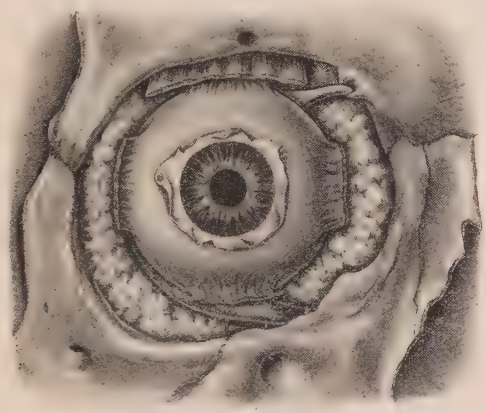

FIG. 4.—The eye muscles seen from in front.

ward just under the roof of the orbit to its insertion into the top of the superior tarsus by a fan-shaped aponeurosis, which is as broad as the lid itself. The motor muscles of the eye are supplied with blood by the muscular branches of the ophthalmic artery. The external rectus muscle is supplied by the *sixth* nerve, the superior oblique by the *fourth* nerve, and the four remaining motor muscles, as well as the levator palpebrae superioris, by the *third* nerve.

THE LIDS.

Under the skin of the lids is a thin layer of connective tissue, and under this the fibers of the *orbicularis muscle.* The orbicularis, which closes the lids, may be divided into

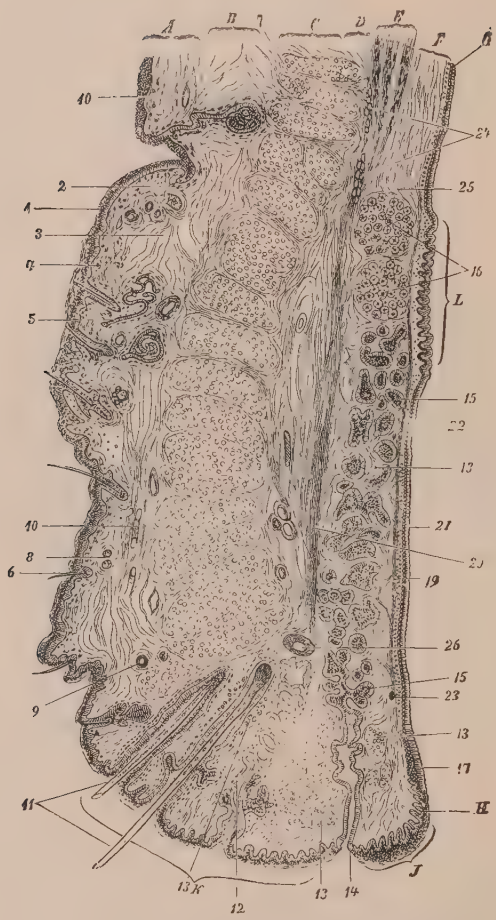

FIG. 5.—Vertical section through the upper eyelid. A, Cutis; 1, Epidermis; 2, Corium; B and 3, Subcutaneous connective-tissue; C and 7, Orbicularis muscle; D, Loose submuscular connective-tissue; E, Insertion of H. Müller's muscle; F, Tarsus; G, Conjunctiva; J, Inner, K, Outer edge of the lid; 4, Pigment cells; 5, Sweat-gland; 6, Hair follicles; 8 and 23, Sections of nerves; 9, Arteries; 10, Veins; 11, Cilia; 12, Modified sweat-glands; 13, Circular muscle of Riolan; 14, Meibomian gland; 15, Section of an acinus of the same; 16, Posterior tarsal glands, submuscular connective-tissue; 21 and 22, Conjunctiva, with its epithelium; 24, Fat; 25, Loosely-woven posterior end of the tarsus; 26, Section of a palpebral artery. (Schafer.)

a palpebral part which lies in the lids proper and an orbital portion which mingles with the muscles of the forehead and cheek. The former arises from the internal palpebral ligament, the latter from the bones in front of the lacrymal groove. The *tensor tarsi,* or Horner's muscle, which is sometimes considered a part of the orbicularis, arises from the lacrymal bone behind the groove. Both of these muscles are supplied by the portio dura of the seventh or facial nerve. Under the orbicularis are *the tarsi,* formerly called cartilage, now known to be dense fibrous tissue. There is one of these thin, flat, elongated plates in each lid to give it form and support, the tarsus of the upper lid being twice as wide as the tarsus of the lower. The tarsi are connected at their extremities and also bound to the subjacent bone by the internal

FIG. 6.—The tarsi. (Schwalbe.)

and external palpebral ligaments. These ligaments are thickened fibers of a circular fascia, the *septum orbitale,* which extends from the rim of the orbit to the orbital edge of the tarsi. Under the tarsi and in groves in their substance are the *Meibomian glands.* They number about thirty in the upper and twenty in the lower lid. They resemble currants on a stem, the stem lying across the tarsus and at right angles to the edge of the lid. Their ducts open on the free margin of the lid. They are sebaceous glands, and the fluid they secrete prevents adhesion of the lid borders. Under the Meibomian glands is the conjunctiva, the mucous membrane which covers the inner surface of the lids. The opening between the lids is called the *palpebral fissure.* The

nasal end of the fissure is the *inner canthus,* the temporal end is the *outer canthus.* The eyelashes are arranged in two rows and their follicles are supplied with sebaceous glands, near which are found the glands of Moll, usually described as modified sweat glands.

LESSON II.

ANATOMY OF THE EYE (*Continued*).

THE LACRYMAL APPARATUS.

The lacrymal apparatus is divided into the secretory part and the excretory part. The former is composed of: 1. The *lacrymal gland*, which is lodged in a fossa at the upper and outer angle of the orbit. It is about the size of the kernel of

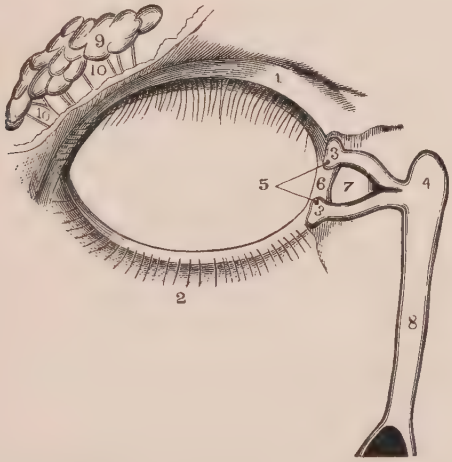

FIG. 7.—Lacrymal apparatus. 1, Upper lid; 2, Lower lid; 3, Canaliculi; 4, Lacrymal sac; 5, Puncta; 6, Plica semilunaris; 7, Caruncle; 8, Nasal duct; 9, Lacrymal gland; 10, Tubules.

an almond, and the fluid it secretes (tears) empties into the conjunctival sac near the superior temporal fornix through small tubes, six to ten in number. 2. A large number of glandular *lobules*, identical in structure and function with the lacrymal gland, imbedded in the loose connective tissue

of the conjunctival fornix, and also found in the tarsi near their orbital borders. These lobules are largest and very much more numerous in the upper lid at the external canthus, which has led to their description in this region as the inferior or palpebral lacrymal gland.

The excretory apparatus begins with the four minute openings: the *puncta,* one of which is located on each lid border about a quarter of an inch from the inner canthus. The puncta open into small tubes, the *canaliculi,* which empty by a common orifice into the side of the *lacrymal sac* at a point just behind the internal palpebral ligament. The sac extends upward two or three millimeters above the internal palpebral ligament and is continuous below with the *nasal duct,* which empties into the inferior nasal fossa. The total length of the sac and duct is about one inch. Their direction is a little backward and outward from the vertical.

THE CONJUNCTIVA.

The conjunctiva is a mucous membrane. Its epithelial layer rests upon the membrana propria which is composed of white fibrous and elastic tissues. The membrana propria is united to the underlying structures by a layer of submucous connective tissue. There is considerable variation in the histology of the three parts which the conjunctiva presents for examination.

1. The *palpebral* conjunctiva lines the inner surface of the lids. It is slightly velvety, due to numerous fine grooves and pits in its surface; the elevations between the grooves are called *papillae.* The membrana propria is closely adherent to the tarsus, and in it are said to be found numerous lymphoid follicles.

2. The *ocular* conjunctiva covers the anterior third of the eyeball except the cornea. Histologically the outer layer of the cornea is similar in structure to that of the scleral con-

junctiva but clinically they appear so different that it is confusing to a student to describe the conjunctiva as covering the cornea. The ocular conjunctiva is very loosely connected to the underlying tissues except at the circumference of the cornea where it adheres firmly. On the ocular conjunctiva, near the inner canthus, is a round, fleshy body, an accumulation of glandular follicles called the *caruncle,* and just external to the caruncle is the *plica semilunaris,* a fold of

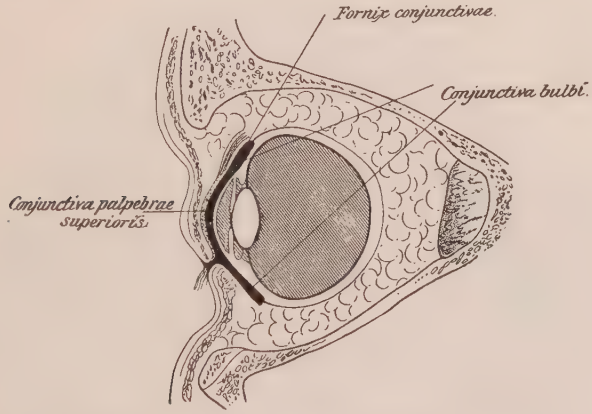

FIG. 8.—Conjunctival sac (in heavy black, exaggerated for sake of clearness.) (After Merkel.)

the conjunctiva which is the analogue of the third eyelid of some animals.

3. The ocular and palpebral portions above described are connected by a loop or fold of mucous membrane called the conjunctival *fornix.* In the fornix are located the glandular lobules which may be considered accessory lacrymal glands.

In the conjunctiva is a considerable plexus of lymphatic vessels, which communicate with the lymph spaces of the cornea. The blood supply is from branches of the ophthalmic, facial and internal maxillary. The sensory nerves come

from the lacrymal and nasal branches of the first division of the fifth nerve.

THE EYEBALL.

The eyeball is a globular body a little less than one inch in diameter. It is not quite a perfect sphere, as the anterior segment, the cornea, has a greater curvature than the rest of the globe. Anatomists are not agreed as to which diameter is the longest.

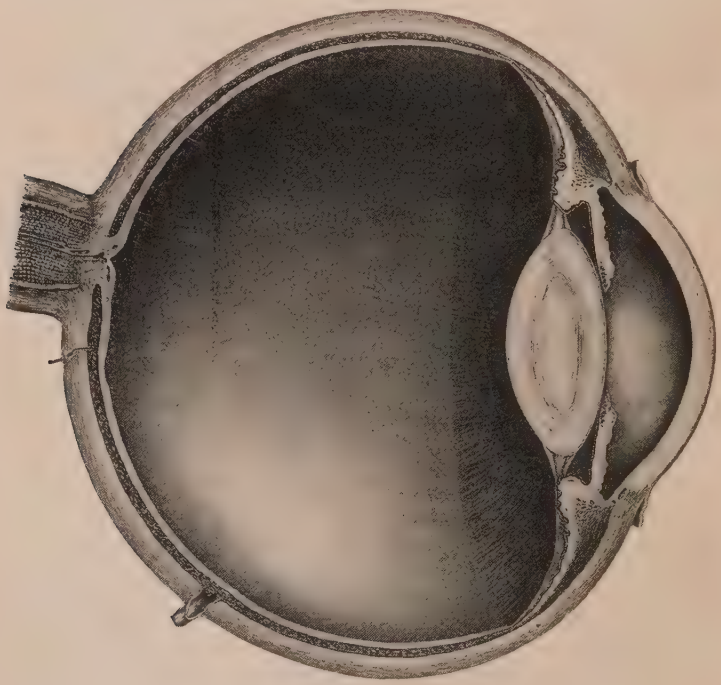

FIG. 9.—A horizontal section of the eyeball. (Deaver.)

The real difference in the size of the eyes of individuals is much less than the apparent difference. The apparent difference being due to the position of the ball in the orbit,

whether set forward or far back, and to the shape of the lids and the width of the palpebral fissure. Normally the eye should be so placed that a line drawn from the upper to the lower orbital margin would just touch the cornea.

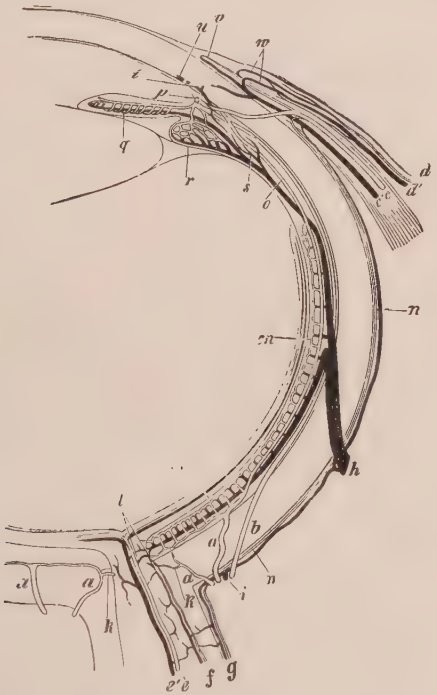

Fig. 10.—Diagram of the blood-vessels of the eye. (Horizontal view; veins black, arteries light, with a double contour.) aa, Short posterior ciliary; b, Long posterior ciliary; cc′, Anterior ciliary artery and vein; dd′, Artery and vein of the conjunctiva; ee′, Central artery and vein of retina; f, Blood-vessels of the inner, and g, of the outer optic sheath; h, Vorticose vein; i, Posterior short ciliary vein confined to the sclerotic; k, Branch of the posterior short ciliary artery to the optic nerve; l, Anastomosis of the choroidal vessels with those of the optic nerve; m, Chorio-capillaris; n, Episcleral branches; o, Recurrent choroidal artery; p, Great circular artery of iris (transverse section); q, Blood-vessels of the iris; r, Ciliary process; s, Branch of a vorticose vein from the ciliary muscle; u, Circular vein; v, Marginal loops of vessels on the cornea; w, Anterior artery and vein of the conjunctiva.

The eyeball consists of three coats or tunics:—

1. The external, fibrous coat, composed of the sclera and cornea.

2. The middle coat, called the uveal tract, composed of the choroid, ciliary body and iris.

3. The nervous coat, the retina.

The interior of the eye is divided by the crystalline lens into the aqueous and vitreous chambers.

The aqueous chamber is divided by the iris into an anterior and posterior chamber.

The following terms are useful for descriptive purposes:—

The *axis* of the eye is a line drawn from the center of the cornea, through the center of the ball, to a point between the optic nerve and macula lutea. The corneal end of this line is the *anterior pole,* the other end, the *posterior pole* of the eye. A circle around the ball at an equal distance from the poles is the *equator.* Other circles around the ball, passing through the poles, are *meridians.*

THE CORNEA.

The cornea is the transparent, glassy-looking, circular membrane which forms the anterior sixth of the eyeball. It has no blood vessels except the capillary loops which encircle its periphery and encroach upon its substance a distance of about one millimeter. The nutrition of the cornea is, in great measure, supplied by lymph derived from this capillary system, though the deeper layers derive some nourishment from the fluid of the anterior chamber by osmosis. The nerve supply is from the fifth and is very profuse.

The cornea has five layers:—

1. In front a layer of *epithelium,* six to eight cells deep, which is continuous with and similar to the epithelium of the scleral conjunctiva.

2. The anterior limiting layer or *membrane of Bowman,* a homogeneous, apparently structureless, resisting layer,

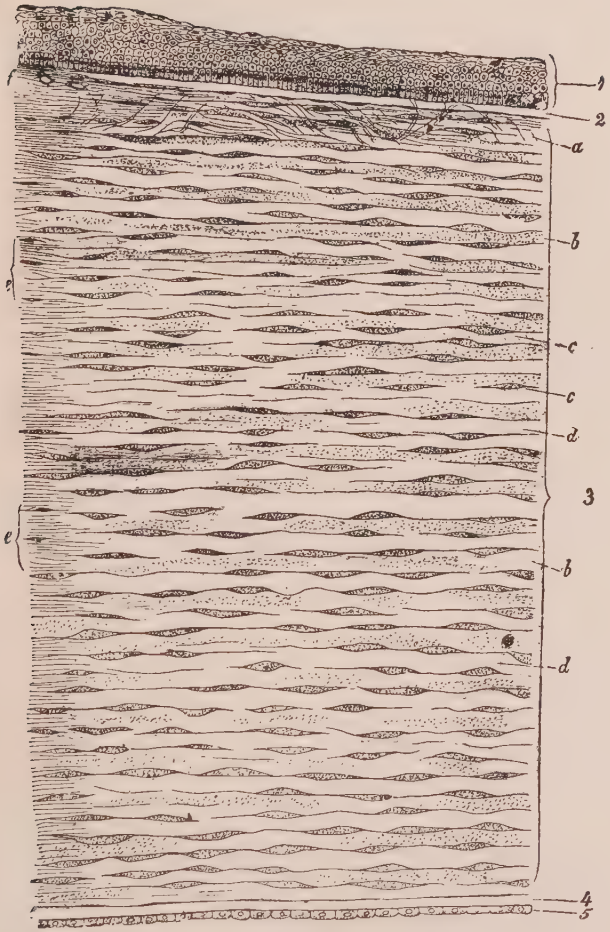

FIG. 11.—Section of cornea near the limbus. (Schafer.) 1, Epithelium; 2, Bowman's membrane; 3, Substantia propria; 4, Descemet's membrane; 5, Endothelium; a, Oblique fibers in the anterior layer of the substantia propria; b, Lamellae the fibers of which are cut across, producing a dotted appearance; c, Corneal corpuscles appearing fusiform in section; d, Lamellae the fibers of which are cut longitudinally; e, Transition to the sclerotic, with more distinct fibrillation, and surmounted by a thicker epithelium; f, Small blood vessel cut across near the margin of the cornea.

which is supposed to be the most instrumental in maintaining the normal corneal curve. It is with difficulty separated from the substantia propria.

3. *Substantia propria* or proper substance of the cornea, a transparent fibrous tissue, not as dense as the preceding, forming the greater part of the thickness of the cornea. It is composed of about sixty layers, the fibers in each running in the opposite direction to the fibers in the layer above and below. Between these layers are found cell spaces which communicate with each other and serve as lymph channels. Within the cell spaces are found the corpuscles, the connective tissue cells of the cornea.

4. Posterior limiting layer or *membrane of Descemet,* a thin, homogeneous, brittle layer, the most resisting of the cornea.

5. A single layer of *endothelial* cells. This layer covers the ligamentum pectinatum at the angle of the anterior chamber, and is continued over the anterior surface of the iris.

LESSON III.

ANATOMY OF THE EYE (*Continued*).

THE SCLERA.

The sclera composes five-sixths of the outer tunic of the eyeball. It is made of white fibrous and yellow elastic tissue with some pigment in its deeper layers. It is essentially of the same constituents as the cornea, but its tissues are so arranged as to almost wholly intercept rays of light. Though the sclera is tough and resisting, the form of the eye is not maintained by it, as it will collapse if the contents of the ball escape.

The sclera is covered by a thin layer of loose connective tissue called the *episclera.* The optic nerve passes through the sclera at a point about 3 millimeters toward the nasal side and 1 millimeter below the posterior pole of the ball. It does not enter in one bundle but divides and passes through numerous openings. This sieve-like part of the sclera is called the *lamina cribrosa.* Around the lamina cribrosa the numerous posterior ciliary vessels and nerves pierce the sclera. Behind the equator the 4 to 6 venae vorticosae find their exit, and about 2 millimeters from the cornea the 5 to 8 anterior ciliary arteries pass in to their distribution to the iris and ciliary body. The sclera and underlying choroid are connected by a very loose, pigmented connective tissue whose meshes communicate so as to form a lymph space: the *perichoroidal.* If the sclera is separated from the choroid half of the brown, pigmented connective tissue adheres to the sclera forming its inner layer, called the *lamina fusca.* The contents of the perichoroidal space escape through lymph channels which are found around the vessels and

nerves that pierce the sclera. The sclera is poorly supplied with blood vessels, its nourishment coming mostly from the lymph of the perichoroidal and periscleral spaces. Completely encircling the cornea, but lying in the scleral tissue, is found the *canal of Schlemm*. There is yet a division of opinion as to whether it is a venous or lymphatic channel. That it is instrumental in draining the anterior chamber is generally accepted.

THE IRIS.

From a point corresponding to the internal junction of the cornea and sclera a curtain is suspended in the aqueous cham-

FIG. 12.—Arteries of the iris. (Sappey.) 1, 1, Long posterior ciliary arteries; 2, 3, Their branches of bifurcation; 4, Recurrent arteries destined for the choroid; 5, 5, 6, 6, Anterior ciliary arteries anastomosing with the long ciliary to form the greater arterial circle of the iris; 7, The lesser arterial circle of the iris.

ber. This curtain, the iris, is the most anterior portion of the uveal tract or vascular coat of the eye. It is composed of muscular fibers, pigment, blood vessels, nerves and connective tissue. The amount of pigment in the iris determines its color, which may vary from the pink of an albino to the

deep brown of a negro. In the center of the iris is a round opening: the *pupil*. The muscular fibers of the iris are involuntary (unstriped) and are divided into radiating and circular fibers. The latter are arranged around the pupil and act as a sphincter in contracting it. These fibers are controlled by the *third* nerve. The radiating fibers are supposed

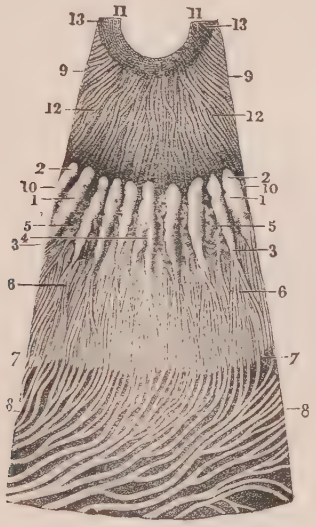

FIG. 13.—Section of ciliary body and iris. 1, 1, Ciliary processes; 2, 2, Their base or rounded extremity; 3, 3, Their apex; 4, A process with bifurcated extremity; 5, 5, Reticulated folds, or ciliary process of the second order; 6, 6, Venules that emanate from these; 7, 7, Ora serrata of choroid; 8, 8, Veins of choroid; 9, Section of iris; 10, 10, Circumference of iris; 11, 11, Small ring of iris; 12, 12, Large ring of iris.

to dilate the pupil and are controlled by the *sympathetic* nerve. The function of the iris is to regulate the amount of light entering the eye. In accomplishing this its action is reflex, the afferent nerve being the optic, the efferent nerve the third. In front of the iris and between it and the cornea is the *anterior chamber*. The region of the anterior chamber

where the cornea and iris unite is called the angle of the anterior chamber. In the angle of the anterior chamber is found the *pectinate ligament,* composed of interlacing trabeculae, which extend from Descemet's membrane of the cornea to the iris. The sponge-like framework of the pectinate ligament incloses numerous intercommunicating spaces: *the spaces of Fontana.* Behind the iris is the *posterior chamber.* Viewed laterally (meridional section), the posterior chamber is triangular in shape. The base of the triangle is the ciliary body; the two sides, the iris and lens; the apex, the point where the pupillary margin of the iris comes in contact with the lens. The anterior and posterior chambers form the *aqueous chamber.* The long ciliary arteries, two in number, arise from the ophthalmic and pierce the sclera, one on each side of the optic nerve. They pass forward between the choroid and sclera to the periphery of the iris, where they divide into an ascending and descending branch. The six to eight anterior ciliary arteries are derived from either the muscular or lacrymal branches of the ophthalmic and pierce the sclera near the corneal junction. They anastomose with the branches of the long ciliary to form the *circulus arteriosus iridis major,* from which branches radiate toward the pupil and around its margin form the *circulus arteriosus iridis minor* (Fig. 12).

THE CILIARY BODY.

The ciliary body lies between the iris and the anterior end of the retina. It is firmly adherent to the sclera at the anterior end but loosely attached behind. It is divided into two parts:—

1. The *vascular* part, which is composed of convoluted blood vessels, connective tissue and pigment, lies next to the vitreous and supplies it and the lens with much of their nourishment. It is also supposed to be the principal agent in the secretion of the aqueous humor. The anterior part of

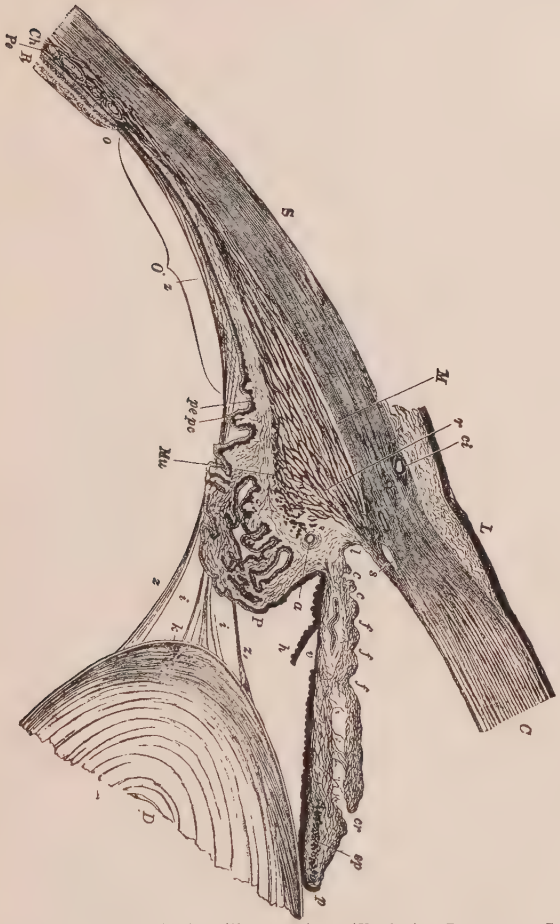

FIG. 14.—Section through the ciliary region. (Fuchs.) C, cornea; S, sclera; Ch, choroid; R, retina; Pe, its pigmented epithelium; o, orra serrata; O, pars ciliaris retinae; this is continued over the ciliary processes; pe, pc, pigmented and non-pigmented cells of pars ciliaris; D, lens; M, ciliary muscle; r, its radiating fibers; Mu, circular fibers; ci, anterior ciliary artery; S, canal of Schlemm; z, origin of ciliary muscle; c, c, f, f, folds and depressions in anterior surface of iris; cr, a crevice in this surface (? artificial); sp, sphincter pupillae; p, edge of pupil; P, most prominent part of ciliary process; h, pigment behind iris, detached at v; a, blood vessel; z, zonula of Zinn; z', z', its continuation or the suspensory ligament; i, i, spaces between the fibers of the suspensory ligament; k, capsule of lens.

the vascular portion is thrown into seventy or eighty projecting tips, the *ciliary processes.*

2. The *muscular* part lies next to the sclera and is the agent of accommodation. Its fibers are unstriped and are arranged in two sets. Those nearest to the sclera run meridionally and those next to the iris equatorially. Contraction of the ciliary muscle is produced by those fibers of the ciliary

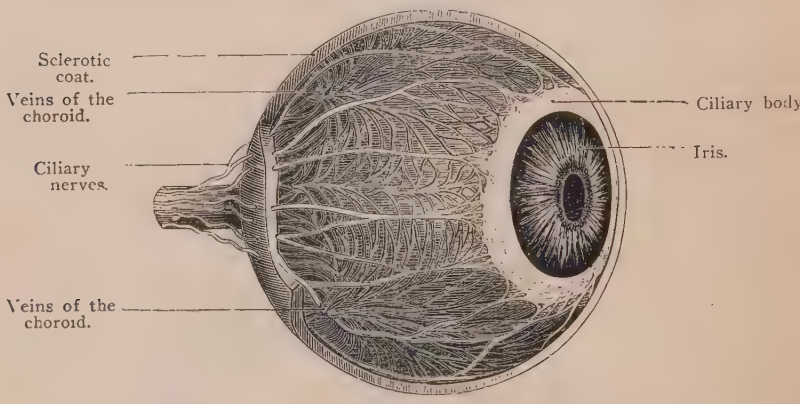

FIG. 15.—Sclerotic coat removed to show the choroid, ciliary muscle, and nerves.

nerves which are derived from the third. The long ciliary nerves, two or three in number, are given off from the nasal nerve which is a branch of the ophthalmic; the ophthalmic being the first division of the trigeminus or fifth. The short ciliary nerves, ten or twelve in number, arise from the *ciliary* or *lenticular ganglion.* This ganglion, which is about the size of a pin head, is found back of the globe between the optic nerve and external rectus muscle. It is supplied by three roots from the fifth, third, and sympathetic nerves. The long and short ciliary nerves pierce the sclera around the optic nerve and pass forward between the choroid and the sclera to the ciliary muscle and iris (Fig. 15).

THE CHOROID.

Extending from the ciliary body back to the optic nerve and lying next to the sclera is a vascular membrane, the choroid. It is connected to the sclera by the loose fibrous trabeculae described on page 17. It has four layers.

1. When the sclera and choroid are torn apart half of this fluffy, pigmented membrane adheres to the sclera (la-

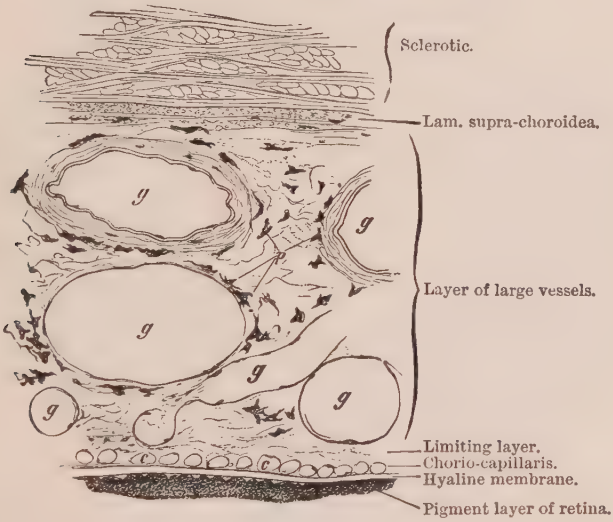

FIG. 16.—Vertical section of the choroid and a part of the sclerotic. g, Large blood-vessels; p, Pigment cells; c, Section of capillaries.

mina fusca) and half to the choroid. The part adhering to the choroid is called the *suprachoroidea.* It is reddish-brown in color, due to the presence of numerous stellate, pigmented cells.

2. Under the suprachoroidea is found the *lamina vasculosa,* a layer of arteries and veins held together by connective tissue. There are also some stellate pigment cells

in this layer. The arteries are derived from the twelve to twenty short posterior ciliary, which arise from the ophthalmic and pass through the sclera around the optic nerve. Some recurrent branches from the long ciliary and the an-

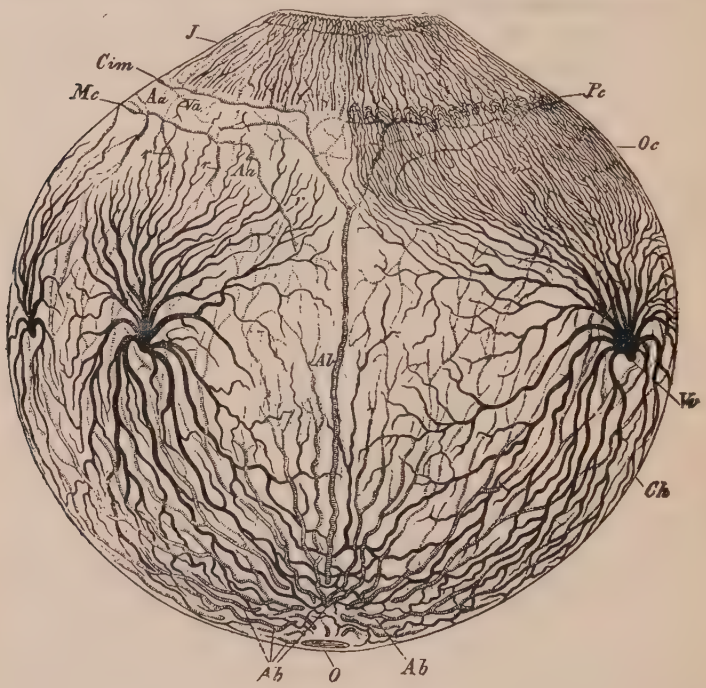

FIG. 17.—Circulation of the choroid (Leber.) O, Optic nerve entrance; Oc, Ciliary region; Pc, Ciliary processes; J, Iris; Aa, Anterior ciliary arteries; Ab, Short posterior ciliary arteries; Al, Long posterior ciliary; Cim, circulus arteriosus iridis major; Mc, Arteries of ciliary muscle; Vv, vena vorticosa.

terior ciliary arteries enter into the anterior portion of the lamina vasculosa.

3. The arteries divide and anastomose to form a capillary layer under the lamina vasculosa called the *chorio-capillaris*. The chorio-capillaris helps to nourish the retina and vitreous.

The veins of the choroid arise from the chorio-capillaris and from the ciliary body and iris and unite into four or six groups. Each group empties near the equator through one vein, the *vena vorticosa.* The venae vorticosae empty into the ophthalmic vein.

4. Under the chorio-capillaris and firmly united to it is found the innermost layer, the *lamina basilis.* It is a thin transparent layer of condensed connective tissue.

LESSON IV.

ANATOMY OF THE EYE (*Continued*).

THE LENS.

Behind the iris and in contact with its pupillary margin lies the crystalline lens, a circular, biconvex, transparent body, composed largely of albumen and water. Its posterior

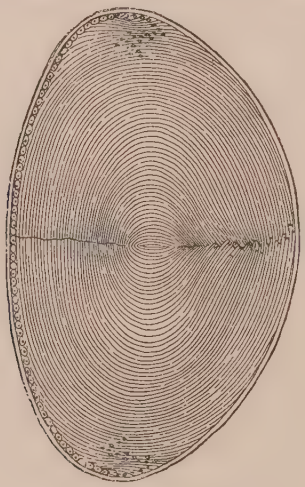

FIG. 18.—Meridional section through the crystalline lens. (Babuchin.)

surface fits into the hyaloid fossa of the vitreous. The curvature of the posterior surface is much greater than that of the anterior. The lens is contained in a transparent capsule, named according to location the *anterior* and *posterior* capsules. The peripheral edge, or the point of union between the anterior and posterior capsules, is called the *equator* of the lens.

The lens is held in position by the suspensory ligament, or *zonula of Zinn,* which is composed of delicate fibers that arise from the posterior surface of the ciliary processes. These fibers are inserted into the equatorial region, some going to the anterior and some to the posterior capsule. The lens is divided into a dense central part, the *nucleus* and a softer peripheral part, the *cortex.* There is no abrupt transition between these parts, there being a gradual centrifugal increase in the density of the lens as age advances.

THE VITREOUS.

The interior of the eyeball, back of the lens, is filled by a transparent, jelly-like connective tissue, the vitreous, which maintains the shape of the eye, and holds the retina and lens in position. It has no blood vessels or nerves and is nourished by lymph from the vessels of the ciliary body, retina and choroid. Through its center from the optic disc to the center of the posterior surface of the lens, runs the *hyaloid canal,* or canal of Cloquet, a lymph channel, which is supposed to communicate in front with the aqueous humor and behind with the lymph spaces surrounding the optic nerve. In fetal life it contains the hyaloid artery. The vitreous is contained within a thin capsule, the *hyaloid membrane.* Anteriorly the vitreous presents the *hyaloid fossa* into which the posterior surface of the lens fits.

THE RETINA.

The fibers of the optic nerve pass through the lamina cribrosa and spread between the choroid and vitreous, forming an almost transparent membrane, the retina. It extends forward to a point corresponding with the union of the choroid and ciliary body, terminating in a wavy line called the *ora serrata.* It is composed of ten layers, the most external or one lying next to the choroid, being a pigment layer which does not terminate at the ora serrata but continues

over the ciliary body and posterior surface of the iris to the margin of the pupil.

The other nine layers are composed of very complicated nerve structures and their supporting connective tissue. In the center of the retina is a round area about one twentieth of an inch in diameter, the *macula lutea*, so called from the yellow color it assumes soon after death. In the center of the macula is a slight depression which appears as a more

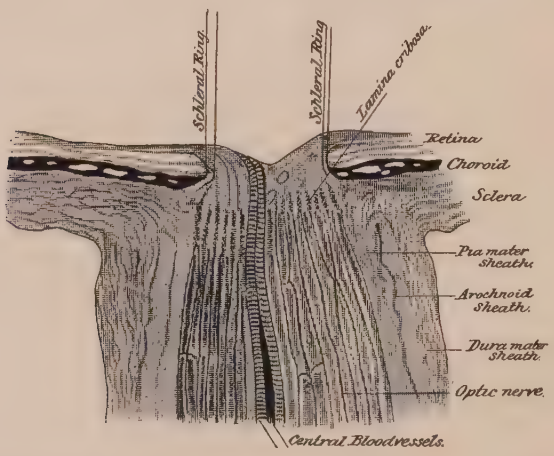

Fig. 19.—Section through optic nerve and papilla. (After Flemming.)

deeply colored point or spot. This spot, which corresponds to the posterior end of the visual axis, is called the *fovea centralis*. It is the center of direct vision and the most sensitive part of the retina. A little toward the nasal side of the retina is seen the large, circular white spot, the *optic disc* or intra-ocular end of the optic nerve. The arteria centralis retinae pierces the optic nerve 1.5 centimeters from the eyeball and passes forward through its center to the optic disc where it divides into its branches, which spread out in all directions in the retina. There is no anastomosis between the

retinal arteries, hence an obstruction of one of them results in destruction of the area which it nourishes. The retinal veins lie by the side of the arteries. The retina is attached to the underlying structures only at the optic disc and at the ora serrata. Shrinkage of the vitreous is prone to cause separation of the retina from the choroid anywhere between these two points.

THE OPTIC NERVES.

An optic nerve may be divided into three parts: 1. Intra-ocular. 2. Orbital. 3. Intra-cranial.

1. The optic nerve fibers pass through the sclera at the lamina cribrosa and then radiate in every direction to form the retina. That portion of the nerve between the lamina cribrosa and the point where it disperses to form the retina is called the head of the nerve, the *optic disc* or optic papilla. With the ophthalmoscope it shows as a round white spot almost in the center of the posterior wall of the eye. (See colored plate.)

2. The orbital portion of the optic nerve extends from the sclera to the optic foramen. It curves in the shape of an S, which enables the eye to move freely in all directions without subjecting the nerve to undue tension. The nerve fibers, about half a million in number, are collected into numerous bundles which are inclosed in a framework of connective tissue. The sheaths of the optic nerve are three in number, the dura, arachnoid and pia mater, which originate from the same membranes of the brain. Under the dural sheath is a lymph space: the subdural, and under the arachnoid another: the sub-arachnoid space. These spaces communicate with the cerebral spaces of the same name. Anteriorly, the three sheaths merge into the sclera.

3. The intra-cranial portion of an optic nerve extends from the optic foramen to the chiasm, a distance of less than one centimeter. In this region it has lost its two outer

sheaths which have merged into the corresponding membranes of the brain. Though the optic nerves proper end at the chiasm, their fibers are conveyed from the chiasm to their termination in the brain by two nerve bundles called the

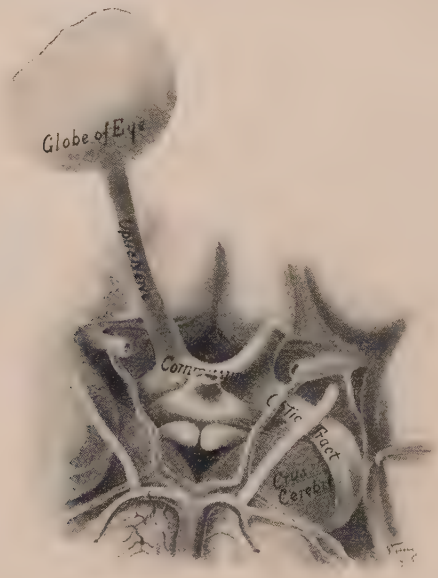

Fig. 20.—Optic tracts and commissure.

optic tracts. In the chiasm or *optic commissure,* which lies in the optic groove of the body of the sphenoid bone, a partial decussation of the optic nerve fibers takes place. The fibers from the nasal side of each retina cross to the optic tract of the opposite side. The fibers from the temporal side of each retina pass back to the brain without decussation. Thus it will be seen that the right optic tract is made up of the fibers which supply the right side of each retina and the

left optic tract the fibers of the left side of each retina (Fig. 21). The principal termination of the optic tracts is in the *cuneus* of the occipital lobe. A small bundle is sent to the nucleus of the third nerve.

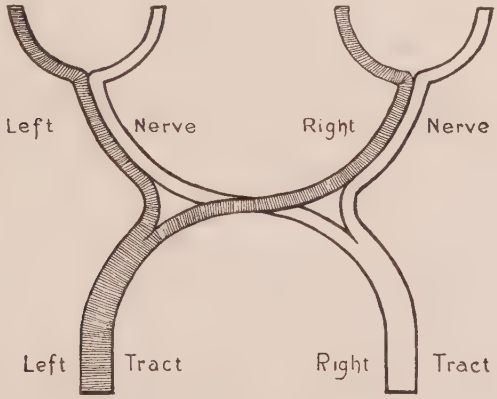

FIG. 21.—Decussation of the optic nerve fibers.

LESSON V.

OPTICS.

LIGHT.

Light is emitted from every point of a luminous body, in successive waves, like the circles which form when a stone is dropped in still water. The number of waves per second and consequently the wave lengths vary with the nature of the luminous body.

A line from a luminous point, perpendicular to a wave front represents a ray of light. It is the smallest sub-division of light traveling in a straight line (Fig 22).

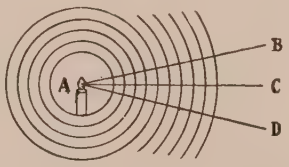

FIG. 22.—The circles represent the light waves emitted from the candle A. AB, AC, and AD perpendicular to the wave fronts represent rays of light from the candle A.

The velocity of a ray of light is less in a dense than in a rare medium. All transparent solids and liquids are denser than air. This change in the velocity of light through different media causes refraction (Fig. 23).

Refraction is the change which takes place in the direction of rays of light when they pass *obliquely* from one transparent medium into another of different density. Rays which pass into the second medium perpendicular to its surface are not deviated (*a a,* Fig. 24).

A ray of light passing from a rarer into a denser medium is bent toward the perpendicular. A ray of light passing

from a denser into a rarer medium is bent from the perpendicular (Fig. 24). The degree of the deviation depends upon the difference in the density of the two media.

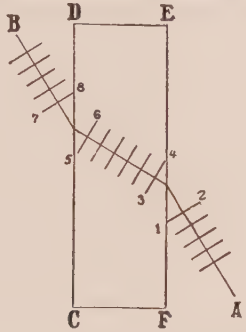

FIG. 23.—AB represents a ray of light passing through a rectangle of glass D E C F. The parallel cross lines represent the series of wave fronts by which the ray is propagated. These wave fronts are such small arcs of a circle that they may be regarded as straight lines. Every part of a wave moves with equal velocity while in the same medium. The point 1 of the wave 1, 2, enters the glass first and its velocity lessens while the velocity of the point 2 remains the same. The result is that point 2 will reach point 4 while point 1 is going to point 3 and the direction of the wave front is changed. Since the ray is perpendicular to the wave fronts its direction is also altered. The same theory will explain the change in the direction of the ray upon emerging from the surface D C.

From Fig. 23 we can also learn why a ray which passes perpendicularly from one medium into another of different

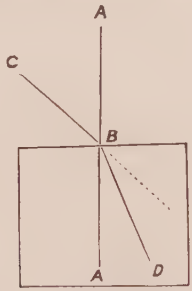

FIG. 24.—AA, perpendicular to surface between air and glass. C B D, ray bent toward perpendicular when passing from rarer medium air into denser medium glass. Reverse the direction and D B C is a ray bent from the perpendicular when passing from the denser medium glass into the rarer medium air.

density is not refracted. If the ray is perpendicular to the surface of the second medium the wave fronts will be parallel with this surface and every part of the wave fronts will strike the second medium at the same time. Though the velocity of the ray will be altered the direction will be the same.

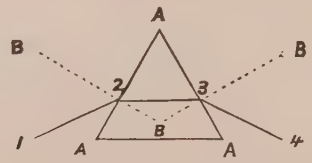

FIG. 25.—A A A, a prism; B B B, perpendiculars to sides of prism; 1, 2, 3, ray bent toward perpendicular when passing from air into glass; 2, 3, 4, ray bent from perpendicular when passing from glass into air.

A refracting prism is any transparent body lying between two plane faces which are not parallel. The apex, sides and base of a prism are terms which explain themselves. The refracting angle is formed by the apex and the two sides. A ray of light upon entering a prism is bent toward the perpendicular, upon emerging it is bent from the perpendicular. A prism always bends rays toward its base (Fig. 25).

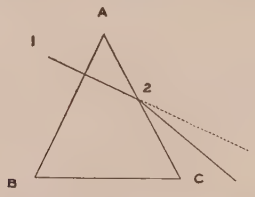

FIG. 26.—The incident ray 1, 2, is perpendicular to the surface A B. It is not refracted until it reaches the surface A C, when it is bent toward the base B C.

If an incident ray is perpendicular to the side of a prism it will not be refracted until it emerges from the other side (Fig. 26).

The image of an object seen through a prism is displaced toward the apex of the prism (Fig. 27).

Formerly prisms were numbered by the degrees of their refracting angles. Now the centrad of Dennett and the prism-diopter of Prentice are more or less complicated methods in vogue. The difference in the results obtained by the

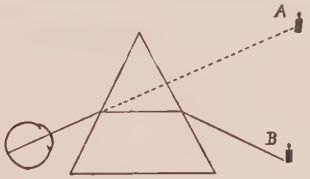

FIG. 27.—The image of the candle B, is displaced toward the apex of the prism and is seen at A.

three methods of computation are not sufficiently vital to warrant their explanation here.

A lens is any transparent medium bounded by two curved surfaces or one plane and the other curved. Lenses are divided into spheric and cylindric.

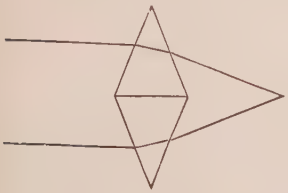

FIG. 28.

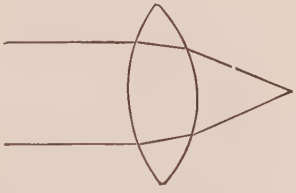

FIG. 29.

A curved surface of a spheric lens is the section of a sphere, hence the name. Spheric lenses are either convex or concave.

A convex lens may be regarded as a series of prisms with their bases directed toward the center (Fig. 28). The strength of these prisms increases from the center toward the periphery in such proportion as to bend all parallel rays passing through them to a common point.

A straight line, perpendicular to both surfaces of a lens, and passing through its center of curvature is called its principal axis. All rays of light parallel with the principal axis

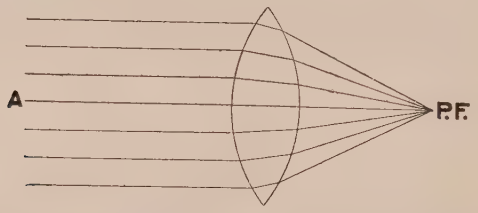

Fig. 30.—The line A through the center of the lens represents the principal axis; P F, the principal focus.

are brought to a focus at a point on the principal axis. This point is called the principal focus. The distance from the optical center of a convex lens to its principal focus is called

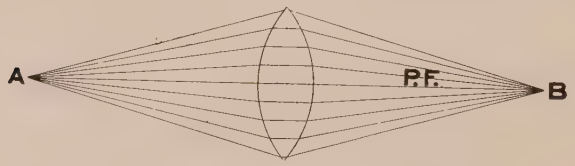

Fig. 31.—P F, principal focus; A B, are conjugate foci.

its focal distance. Rays which diverge from the principal focus and pass through a convex lens are rendered parallel (Fig. 30).

Rays which diverge from a point farther than the principal focus and pass through a convex lens are brought to a focus again. The two points on the axial ray where these rays come together are called conjugate foci (Fig 31).

Rays which diverge from a point nearer than the principal focus remain divergent after passing through a convex lens (Fig. 32).

A concave lens may be regarded as a series of prisms with their bases directed away from the center (Fig. 33),

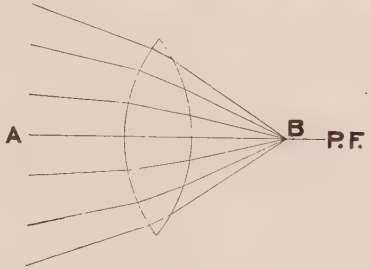

Fig. 32.—P F, principal focus. Rays from the point B, between the lens and its principal focus are rendered divergent.

the strength of these prisms increasing from the center toward the periphery.

Fig. 33.

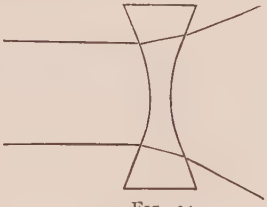

Fig. 34.

All rays of light passing through a concave lens parallel with the axial ray are rendered divergent. If these divergent

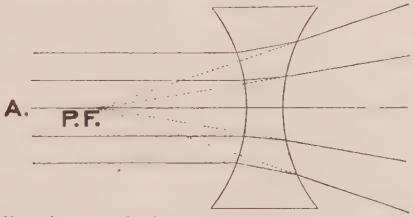

Fig. 35.—The line A through the center of the lens represents the principal axis. P F, the principal focus which is negative or virtual.

rays be projected backward in a straight line they will come to a negative or virtual focus at a point on the axial ray (Fig.

35). The distance from this point to the optical center of the lens is its focal distance. As a concave lens always diverges rays, regardless of the distance from whence they come, its focus is always negative.

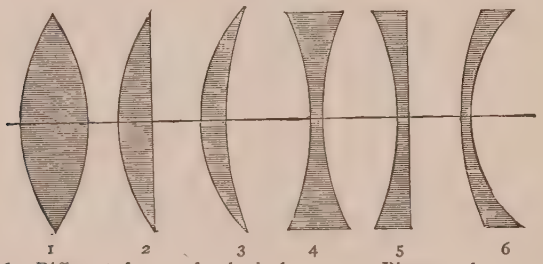

FIG. 36.—Different forms of spheric lenses. 1, Biconvex lens; 2, Planoconvex lens; 3, Concavoconvex, or convergent meniscus; 4, Biconcave; 5, Planoconcave; 6, Convexoconcave, or divergent meniscus.

There are three forms of convex lenses: planoconvex, biconvex and concavoconvex; also three forms of concave lenses: planoconcave, biconcave and convexoconcave (Fig 36).

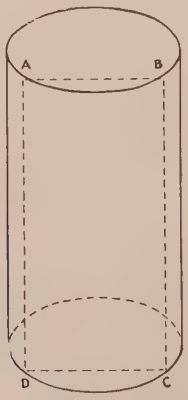

FIG. 37.—A, B, C, D is a section of a cylinder cut parallel to its axis.

Convex lenses are designated + (plus); concave lenses — (minus).

A cylindric lens derives its name from the fact that it is the section of a cylinder (Fig. 37). The section is cut parallel with the axis of the cylinder. The rays which pass through the axis are not refracted since there is no curvature in this direction. Rays passing through the meridian which is perpendicular to the axis undergo the maximum amount of refraction, since the lens presents its greatest degree of curvature in this direction. The refracting value of any

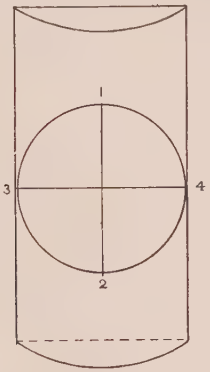

FIG. 38.—A lens cut from a section of a cylinder. The meridian 1, 2, is parallel to the axis of the cylinder; it has no curvature and no refracting power. The meridian, 3, 4, being thicker at the middle than at the ends, is convex; it will bring parallel rays of light to a focus.

meridian lying between these two principal meridians depends upon its proximity to the region of no refraction (the axis), or to the region of maximum refraction (Fig. 38).

Cylindric lenses may be convex or concave (Figs. 39 and 40). They are designated by the abbreviation C., or cyl.

The strength of a lens or its ability to change the direction of rays depends upon the density of the material of which it is made and the degree of curvature of its surfaces.

The greater the strength of a lens the shorter is its focal distance. The term diopter is used in numbering lenses. A lens whose focal distance is one meter is called a one diopter

lens, or 1.D. A lens of two meters focus has only one-half the refractive power of a 1.D lens and is called a half diopter lens, or 0.50 D. If the focal distance is one quarter of a meter it is a 4.D lens, etc.

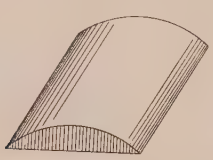

FIG. 39.—A convex cylinder.

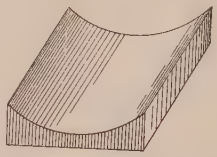

FIG. 40.—A concave cylinder.

LESSON VI.

REFRACTION AND PHYSIOLOGY OF THE EYE.

In order that an eye may receive a distinct visual impression of an object, rays of light from that object must be brought to a focus on its retina. The cornea and crystalline lens act as convex lenses in bringing rays of light which enter the eye to a focus on the retina.

The average width of the pupil is 4 millimeters, and rays which enter it from a point 6 meters (20 feet) distant necessarily diverge very slightly, amounting at the most to only $^4/_{6000}$, therefore in estimating refraction rays from this distance and greater are arbitrarily considered parallel. When from a point under 6 meters they are considered divergent.

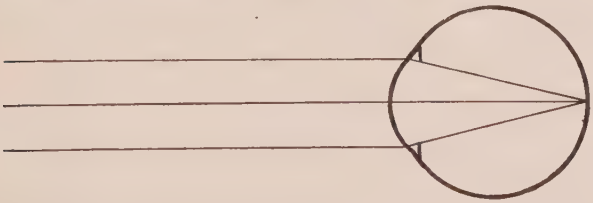

FIG. 41.—Parallel rays of light brought to a focus on the retina as in the emmetropic eye.

EMMETROPIA.

An eye, which in a state of rest brings parallel rays of light to a focus on its retina, has normal refraction and is called emmetropic (Fig. 41). The principal focus of its dioptric system is at its fovea centralis, and rays of light from the fovea, after passing through the lens and cornea will be emitted parallel (Fig. 30). It must be remembered that an

emmetropic eye is not necessarily a normal eye but is an eye with normal refraction.

ACCOMMODATION.

Let the vision of an emmetropic eye be concentrated upon a distant object, for instance a building across the street, then hold a pencil in the line of vision, a foot in front of the eye. The building will be distinctly seen but the image of the pencil, though visible will be blurred. Now if the vision be concentrated upon the pencil its outline will become distinct and the image of the building will be blurred.

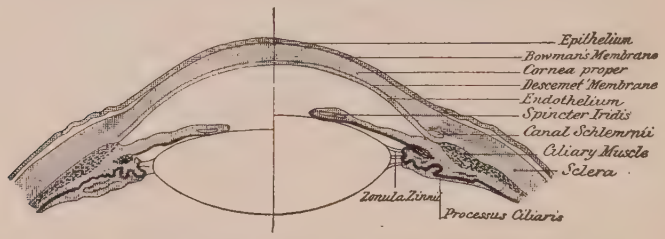

FIG. 42.—The left half represents the eye at rest; the right, during accommodation.

This instantaneous difference in the vision is effected by the power which the eye has of changing its focus for different distances.

We have seen that an emmetropic eye, in a state of rest, will focus upon its retina, all objects at a distance of twenty feet or over; that is, objects which emit parallel rays.

Rays from a near object are divergent, and in order for an emmetropic eye to have distinct vision of a near object it must increase its focal power, as it not only has to focus parallel rays, but has first to make the divergence parallel. This is accomplished by contracting the circular or equatorial fibers of the ciliary muscle. Contraction of the ciliary muscle relaxes the suspensory ligament and capsule of the lens. When the pressure of the capsule is relieved the lens becomes

more convex by an inherent elasticity. Increase in its convexity increases its focusing power. This power the eye possesses of increasing its focal strength is called accommodation (Fig. 42).

PRESBYOPIA.

The elasticity of the crystalline lens diminishes gradually from childhood to old age. Under normal conditions this loss of elasticity is not felt until about the forty-fifth year, but at this period the power of accommodating is so lessened that convex glasses have to be resorted to for near vision. This physiologic loss of accommodative power is called presbyopia. Weakness of the ciliary muscle is also a contributing factor in presbyopia, particularly in old age. As accommodation diminishes the reading glass must be strengthened, necessitating a change about every two years.

CONVERGENCE.

When an eye is directed toward an object so that the image of the thing looked at falls upon the fovea centralis, the eye is said to fix that object. Normally both eyes fix the same object, and in order to do this when it is brought near to the face, both eyes have to turn inward; the nearer the object the more the eyes turn in. The turning in of the eyes necessary to fix near objects is called convergence.

FIELD OF VISION.

When the eye is fixed on an object, other things besides the one looked at are visible. Those nearest the one fixed are most distinct and the greater the distance of an object from the one fixed, the less distinctly is it seen. That area in which objects are visible, the eye being fixed, is the field of vision. It will be seen from Fig. 43 that the nasal field of each eye extends to about 48 degrees from the object looked at, therefore if both eyes look at the same object there is an overlapping of the two fields, or an area that is common to

both eyes. This area which extends to about 48 degrees on each side of the object is called the binocular field of vision.

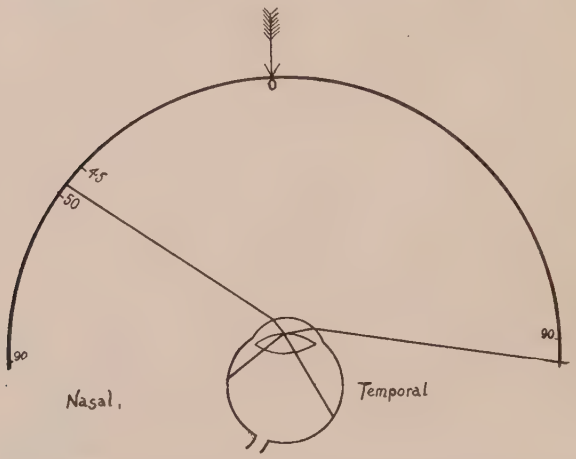

FIG. 43.—Field of vision of a right eye. The arrow at o being fixed (looked at) all objects on the temporal side within the area described by about 95 degrees of a circle are visible; all objects on the nasal side within about 48 degrees are visible. The nasal side of the field is restricted by the bridge of the nose.

COLOR PERCEPTION.

A ray of sunlight passed through a prism and projected upon a screen forms a band of colors ranging from red to violet. The red is toward the apex and the violet toward the base of the prism. Between the red and violet there are gradations of orange, yellow, green and blue. The wave lengths of these colored rays gradually decrease from the red rays which are .000760 mm., to the violet, which are .000397 mm. The greater the wave length of a ray of light, the less it is deviated by passing through a medium of different density, hence the power of a prism to separate a ray of white light into its elements. The six colors of the solar spectrum, red, orange, yellow, green, blue and violet, are called *simple*

colors because it is found by passing any one of them through a prism that no further disintegration takes place (Fig. 44). Red, green and violet can be mixed to produce any of the other colors, but as no combination can produce either of these three they are called the *primary colors.*

Many theories have been offered to explain the phenomenon of color perception, but none has yet supplanted the Young-Helmholtz. This is that we have three primary color

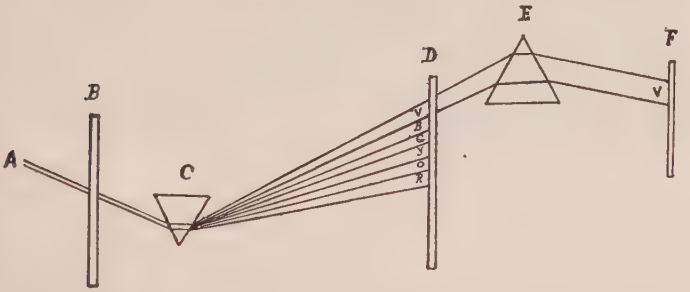

FIG. 44.—B is a screen which intercepts all rays of light except the ray A. The prism C separates the ray A into the simple colors, red, orange, yellow, green, blue and violet, which are thrown on the screen D. The violet rays, if passed through the screen D, and prism E, would show on the screen F, as violet; no further disintegration taking place.

perceptions corresponding to the three primary colors of nature, and that there are red perceptive fibers, green perceptive fibers and violet perceptive fibers in our retinas. These different nerve fibers are stimulated by light waves of different lengths. Equal stimulation of all three produces the sensation of white, and just as all the colors in nature can be produced by mixing the spectrum red, green and violet, so can every color sensation be produced by stimulation of the red, green and violet perceptive fibers in varying proportions. The absence or impairment of one or more of the primary perceptions constitutes *color-blindness;* the characteristic of the defect depending upon which element is missing or impaired. The condition is congenital, does not disturb vision,

is not dependent upon any demonstrable pathologic lesion, is irremediable and is often hereditary. There are other forms of color-blindness depending upon diseases of the retina and optic nerve, which will be described in connection with those diseases.

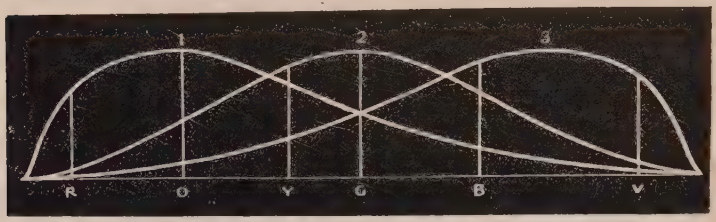

FIG. 45.—A diagram of color perception. 1, Red; 2, Green; 3, Violet. The height of the curve from the base line indicates the proportions in which the primary colors are mixed to produce the simple colors of the spectrum, red, orange, yellow, green, blue and violet.

LESSON VII.

ERRORS OF REFRACTION.

AMETROPIA.

Any variation from the state of normal refraction or emmetropia is called ametropia. Ametropia appears in three forms: hyperopia, myopia and astigmatism. Presbyopia is not considered a form of ametropia as it is a physiological change which overtakes the emmetropic as well as the ametropic eye.

HYPEROPIA.

If the focus of parallel rays is at an imaginary point behind the retina, the eye being at rest (*i. e.,* not accommodating), it is far-sighted or hyperopic. Hyperopia is due to shortness of the antero-posterior axis of the eyeball or to lack of sufficient focal strength in the cornea and lens (Fig. 46).

As the fovea centralis lies between the dioptric media of the eye and their principal focus, rays of light from the fovea, after passing through the lens and cornea, will be divergent (Fig. 32). If rays from the fovea are divergent when they leave an eye only rays similarly convergent upon entering it will be brought to a focus on the fovea. There are no convergent rays in nature, for as we have already learned those from 20 feet or more are parallel and those from a point less than 20 feet are divergent.

There are but two ways of converging the rays of nature so as to focus them upon the retina of the hyperopic eye. The eye must increase its focal strength or rays must be artifically converged before entering it.

By accommodating the lens can increase its focal strength, therefore hyperopic eyes accommodate constantly for distant

vision and for near vision must add the amount of accommodation exercised for distance to the amount it would accommodate if it were emmetropic. The continuous contraction of the circular fibers of the ciliary muscle required by the accommodative effort of the hyperopic eye causes a number

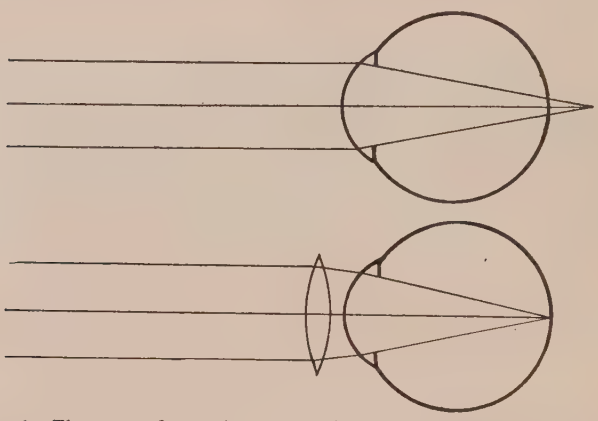

Fig. 46.—The upper figure shows parallel rays of light focused behind the retina as in hyperopia. The lower figure shows the influence of a convex lens in bringing the focus to the retina.

of symptoms, the most prominent of which is headache. See asthenopia, page 62.

Rays may be artificially converged before entering a hyperopic eye by the use of a convex lens, and thus brought to a focus on its fovea centralis without any accommodative effort (Fig. 46).

MYOPIA.

If the focus of parallel rays is at a point in front of the retina, the eye being at rest, it is near-sighted or myopic. Myopia is due to too great length of the antero-posterior axis of the eyeball, or to too great focal strength of the cornea and lens (Fig. 47). As the fovea centralis lies farther from the dioptric media of the eye than their principal focus, rays of

light from the fovea, after passing through the lens and cornea, will be convergent and will come to a focus in front of the eye (Fig. 31). If rays from the fovea are convergent when they leave an eye only rays similarly divergent upon entering it will be brought to a focus on the fovea. Only

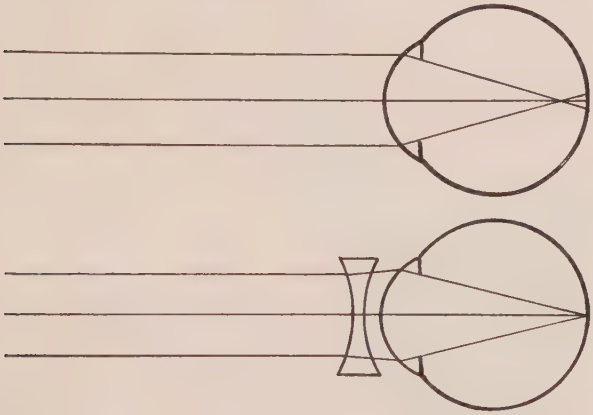

FIG. 47.—The upper figure shows parallel rays of light focused in front of the retina as in myopia. The lower figure shows the influence of a concave lens in moving the focus back to the retina.

rays from an object less than 20 feet are divergent, hence distance objects are seen poorly by a myopic eye and if its error is pronounced they are not seen at all.

There is no way of decreasing the focal strength of the lens, therefore no effort on the part of a myope can overcome his defect. It can be overcome, however, by artificially diverging rays before they enter the eye. This is accomplished by the use of concave lenses (Fig. 47).

The myopic eye is adjusted for near vision since only divergent rays can be focussed on its retina and rays from near objects only are divergent. Hence the term near sight.

The greater the degree of myopia the greater the degree of divergence which rays must have in order to focus on the

fovea and consequently the nearer an object must be brought to the eye.

In hyperopia the effort of accommodation necessitates the constant exercise of the circular fibers of the ciliary muscle, and we find in hyperopic eyes that the circular fibers (Müller's muscle) are increased in size and number as in Fig. 48.

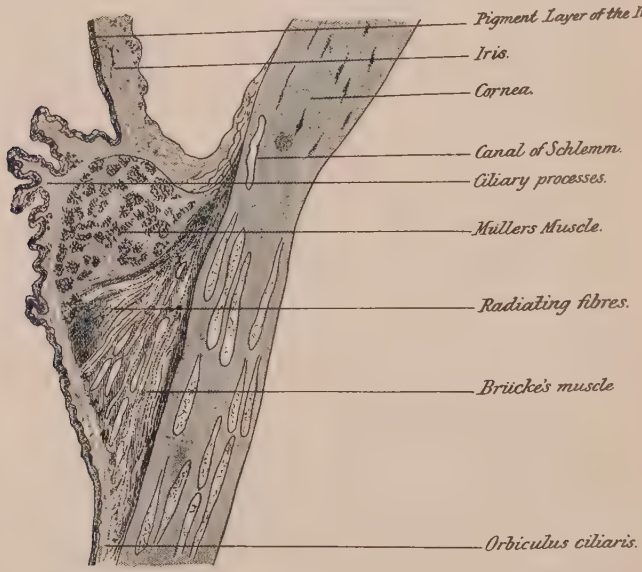

FIG. 48.—Ciliary body of a hyperopic eye. (After Iwanoff.)

Accommodation would make the vision of a myopic eye worse, and we find in these eyes that the circular fibers of the ciliary muscle are small in size and number.

In myopia of high degree, when the vision of the patient is not sufficient for his needs and no condition exists contraindicating the operation, removal of the crystalline lens may be resorted to. This procedure has often given most gratifying results.

It will be seen from what has preceded that (without lenses) the emmetropic eye sees distant objects in a state of rest (without accommodating) and must accommodate for near objects; the hyperopic eye must accommodate to see both distant and near objects; and the myopic eye sees distant objects imperfectly or not at all and near ones without accommodating unless the myopia is of low degree.

ASTIGMATISM.

In emmetropia, hyperopia and myopia, the curvature of the cornea is the same in every meridian, and its refracting power is the same through every part, vertical, horizontal and

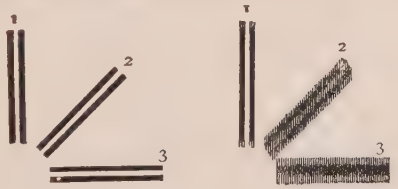

FIG. 49.—Appearance of lines running in different directions. A, as seen by the normal eye; B, as seen by the astigmatic eye. (Jackson.)

oblique. All rays that enter these eyes are brought to a common focus. Sometimes the cornea has meridians of different curvature producing greater refraction in some meridians than in others. Such a condition constitutes astigmatism. In astigmatism the rays passing through the meridian of greatest refraction reach their focus nearest the cornea, while those passing through the least refracting meridian come to a focus farthest back. The meridians of highest and lowest refracting power are at right angles to each other and are called the *principal meridians.* Astigmatism is sometimes due to unequal curvature of the meridians of the crystalline lens or to an oblique position of the lens with regard to rays entering the pupil. Astigmatism may be simple, compound or mixed. When one of the principal meridians is emmetropic and the other hyperopic or myopic, the astig-

matism is simple. When both principal meridians are hyperopic but one more so than the other, or both principal meridians are myopic, one more so than the other, the astigmatism is compound. When one principal meridian is hyperopic and the other myopic the astigmatism is mixed.

The inequality of the refractive power of the principal meridians in an astigmatic eye can only be equalized by the use of a lens which has different refractive power in its principal meridians. This requisite is found in the cylindric lens. A cylindric lens alone will correct simple astigmatism; for compound and mixed astigmatism a combination of cylindric and spheric lenses is necessary.

ISOMETROPIA, ANISOMETROPIA AND ANTIMETROPIA.

When the refractive condition of a pair of eyes is the same, or if there is any difference in them, it is too small to be detected by our present methods, they are said to be isometropic.

If the refractive condition of the two eyes is the same in kind but different in degree they are said to be anisometropic. A slight difference in them is very common and can hardly be considered a pathologic state. The term anisometropic is generally used when the difference is sufficient to impair vision or cause disturbance of the nervous system.

If the refractive condition of the two eyes is different in kind they are said to be antimetropic. The term antimetropia does not refer to the degree of error, which may be equal or unequal.

LESSON VIII.

DISEASES OF THE OCULAR MUSCLES.

STRABISMUS.

Normally both eyes fix the same object. The image of the object looked at falls upon the fovea centralis of both eyes. This is accomplished by the co-ordination and association of movement of the six external ocular muscles of each eye. In looking up, down, right or left, the eyes move together and binocular or single vision results, because the images of

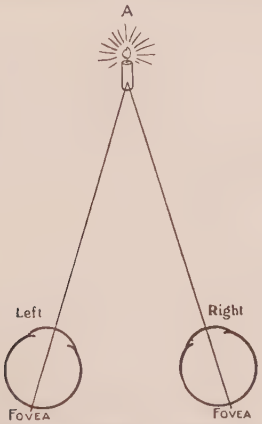

Fig. 50.—The two eyes in a state of muscular equilibrium. The image of the candle A falls upon the fovea centralis of each eye.

objects in the field of vision fall upon *identical parts* of each retina, the upper half of the right retina corresponding to the upper half of the left, the right or temporal side of the right retina to the right or nasal side of the left, etc. In this normal state the eyes are said to be balanced or in equilibrium (Fig. 50).

If this association of movement is disturbed, so that the image of an object falls upon the fovea centralis of one eye and not upon the fovea of the other, we have strabismus, or squint. The eye which receives the image of the object looked at upon its fovea is called the *fixing eye;* the other is called the squinting, or *deviating eye.* The deviation may be in any direction, depending upon which muscle or set of muscles is affected.

Strabismus is either paralytic or concomitant.

PARALYTIC STRABISMUS

Is produced by loss of power in one or more of the ocular muscles. This loss of power may be total (paralysis) or partial (paresis), the latter being by far the more frequent. When the paralysis is confined to those fibers of the third nerve which supply the iris and ciliary body the condition is known as *internal ophthalmoplegia.* When all the muscles of an eye are paralyzed except those of the iris and ciliary body the condition is known as *external ophthalmoplegia.* If both the internal and external ocular muscles are paralyzed the condition is known as *total ophthalmoplegia.*

Symptoms.—1. **Movement of the eye** in the direction of the action of the affected muscle is limited or lost. If an external rectus is paretic its antagonist, the internal rectus, will pull the eye inward. The deviation of the afflicted eye, the sound eye fixing, is called the *primary deviation.* If the sound eye be covered by a card and the paretic eye fixes the object, it will be seen by looking behind the card that the sound eye has now deviated in a direction opposite to the primary deviation, and that the deviation is greater. This is called the *secondary deviation.* In paralytic strabismus the secondary deviation is always greater than the primary, because the same amount of nervous impulse necessary to produce a given result in the weakened muscle is also conveyed to its sound associate and results in its overaction.

2. **Diplopia** or double vision results because the muscular imbalance or lack of equilibrium prevents images of objects in the field of vision from falling upon the indentical parts of each retina. The image of the fixing eye is called the *true image;* the image of the deviating eye the *false image.* If an external rectus is paralyzed the anterior pole turns in, the posterior pole out. The image of the object fixed by the sound eye falls upon the retina of the diseased

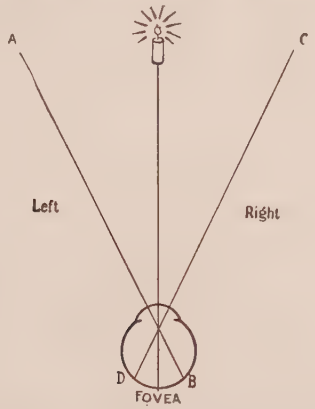

FIG. 51.—The eye is fixed on the candle. Objects in the right field at C are perceived by the left side of the retina at D. Objects at A are perceived by the right retina at B.

eye, to the inner side of the fovea centralis and is projected to the temporal side of its field. This is due to the fact that the patient is in the habit of locating objects in the temporal field which are perceived by the nasal side of the retina (Fig. 51) and he does not take into consideration the deviation of his eye. If it is an internal rectus that is weakened the eye turns outward and the image of the object fixed by the sound eye falls on the retina of the diseased eye to the outer side of its fovea and is projected to the nasal side of the field.

Thus it will be seen when the strabismus is convergent the image of the right eye is on the right side, the image of

the left eye on the left side. This is called *homonymous diplopia* (Fig. 52).

When the strabismus is divergent the image of the right eye is to the left, the image of the left eye to the right. This is called crossed or *heteronymous diplopia* (Fig. 53). In vertical strabismus the lower image belongs to the eye turned up, the upper image to the eye directed downward.

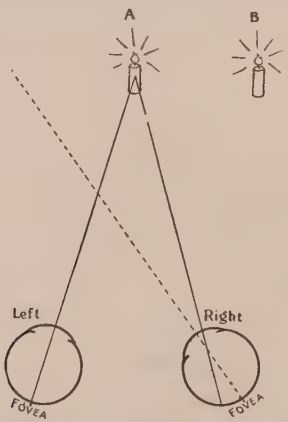

FIG. 52.—Convergent strabismus of the right eye. The image of the candle A, falls on the retina at the inner side of the fovea and is seen at B. Homonymous diplopia. A, true image. B, false image.

3. **Inaccuracy in the determination of the position of objects** in that part of the field toward which the affected muscle normally directs the eye is observed in paralytic strabismus. In looking toward an object on our right side, we determine by experience its distance to the right by the amount of innervation necessary to direct the visual axes toward it. If the right external rectus muscle is paretic an unusual amount of energy is necessary to fix the object and it will seem farther toward the right side than it really is.

4. **Vertigo, nausea and headache** are troublesome features of paralytic strabismus. The vertigo is produced by

diplopia and by the inability to properly locate objects in part of the field of vision.

5. **A peculiar carriage of the head** will be observed. It will be turned in such a way as to overcome the diplopia by excluding the paretic muscle. If the right external rectus muscle is weakened the face will be turned toward the right

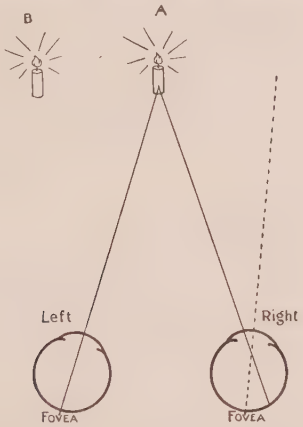

FIG. 53.—Divergent strabismus of the right eye. The image of the candle A, falls on the retina at the outer side of the fovea and is seen at B. Heteronymous diplopia. A, true image. B, false image.

side, which is equivalent to directing the gaze to the left, in which act the paretic muscle would have to take no part.

Cause.—The cause may be found in the brain, the nerve-trunk, or in the muscle itself. If the lesion is in the brain it is said to be *central;* if in the nerve or its destribution it is *peripheral.* When the lesion is central there will probably be more than one muscle involved, other cerebral symptoms will be present and loss of power in the muscle or muscles will be gradual and progressive. When the lesion is peripheral the paralysis is generally limited to one muscle, there are no other symptoms and the loss of power is more complete. It is due to syphilis in about half of all cases and in the other half to

exposure to cold, rheumatism, diphtheria, tabes dorsalis, diabetes, poisons, tumors, meningitis, aneurism, periostitis, hemorrhage, wounds, fractures, and hysteria.

Treatment.—The subjective symptoms can be relieved at once by covering the diseased eye. If the patient wears glasses, a ground glass on that side is effective. Treatment of paralytic strabismus must depend upon the cause. When due to syphilis or rheumatism, the remedies appropriate to these conditions are indicated. When the result of debilitating causes, such as diphtheria, chronic poisoning, etc., give general tonics including strychnin. A weak current of electricity may be tried, the positive pole being placed over the affected muscle, the negative pole on the back of the neck. When the deviation is slight and has become fixed, relief is often afforded by wearing prisms. In selected cases operative treatment, tenotomy or advancement, may be helpful.

LESSON IX.

DISEASES OF THE OCULAR MUSCLES (*Continued*).

CONCOMITANT STRABISMUS.

Symptoms.—There is an absence of the normal association of movement of the ocular muscles without loss of their power. The muscles have their normal strength, but they do not work together so that each eye will fix the same object

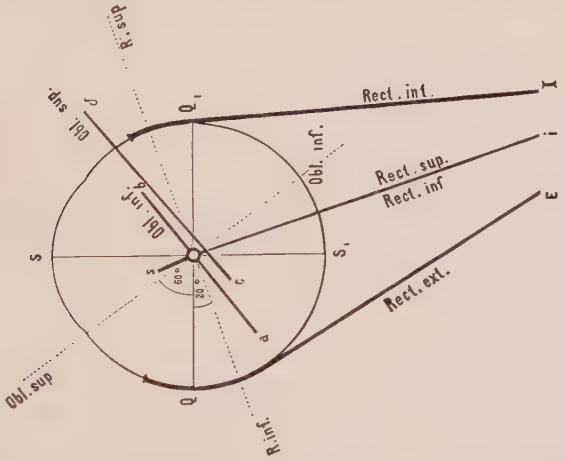

FIG. 54.—Scheme of the action of the ocular muscles. Q E, Direction of traction of ext. rect.; Q,, I, Of int. rect.; S i, Of sup. and inf. recti; a b, Of inf. oblique; c d, Of sup. oblique; O, Point of rotation; Q Q,, Transverse axis.

at the same time. The primary and secondary deviations are equal. The deviation may be *monolateral* or *alternating;* if the latter, vision in each eye will probably be the same. Internal concomitant strabismus is a condition which usually manifests itself early in life, the average age being about three years. Divergent concomitant strabismus usually man-

ifests itself at about the age of puberty or a little later. Diplopia is rare in concomitant strabismus; the squinting eye is often so amblyopic as not to perceive the false image, and if it has good vision, concomitant squint arises so early in life that there is developed, with the growth of the child, a power of the mind to exclude the false image.

Cause.—There is more to discover relative to the cause of concomitant strabismus than has yet been revealed, but the following may be given as etiological factors:—

1. **Hyperopia** exists in three-fourths of all cases of internal concomitant strabismus. Convergence and accommodation are associated actions, and in hyperopia there is excessive accommodation and the associated convergence sometimes amounts to internal strabismus. Relief of the hyperopia by convex glasses will at times straighten these eyes. But that hyperopia is not a prime factor in the production of strabismus is proven by the fact that, in the majority of cases, the correction of the hyperopia by glasses has no effect on the strabismus, and also that there are so many cases of high degree of hyperopia in which there is no strabismus.

2. **Myopia** is associated with divergent strabismus and the explanation is that the convergence necessary to focus at the far point of a myopic eye, which is very close to the face, puts too great a strain on the internal rectus muscles, one of them gives up and divergence results. The exception to this rule is proven by the fact that only a very small proportion of the cases of myopia diverge.

3. **Amblyopia** or poor vision in the squinting eye is found in many cases of strabismus (72 per cent., Nagel) and the weight of authority is in favor of the view that the amblyopia is congenital and is the cause of the strabismus, the stimulation to binocular vision not being present. However, a respectable minority claim the amblyopia to be the effect of

non use of the squinting eye and not the cause of the strabismus. But that congenital amblyopia is only a factor in the production of squint is proven by the absence of squint in the majority of amblyopic eyes.

4. **Unusual Development of a single ocular muscle** is another possible element in the production of concomitant strabismus, the internal rectus being often unnaturally strong in internal strabismus and the same is true of the external rectus when the deviation is outward.

Treatment.—Rarely concomitant strabismus disappears

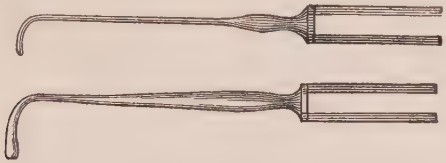

FIG. 55.—Large and small strabismus hooks.

without medical aid. Sometimes it is entirely removed by wearing the glass which corrects the total refractive error, consequently these eyes should be tested and the proper glasses ordered as soon as the child is old enough to wear them. It is also good practice to cover the fixing eye with a bandage for a part of each day to compel the use of the deviating eye.

The treatment for the remaining cases is operative, tenotomy of the over-active muscle or advancement of its antagonist. Operations for strabismus should not be performed before seven or eight years of age, except in rare cases, owing to the tendency to development of over-effect with the growth of the child.

INSUFFICIENCY OF THE OCULAR MUSCLES, HETEROPHORIA OR LATENT SQUINT.

This condition differs from strabismus only in degree, the tendency to deviation being overcome by the desire for

binocular vision. It may be concomitant or paretic. There is lack of balance of the ocular muscles but parallelism of the visual lines is maintained by an unconscious effort on the part of the patient. The effort to maintain this parallelism and secure binocular vision generally results in *asthenopia,* which may be manifested by pain over the insertion of the weak muscle, blurred vision, inability to do close or protracted work, photophobia, subacute congestion of the conjunctiva, headache, vertigo and other neuroses. The following terms were introduced by Stevens to indicate the conditions present in latent squint: *Orthophoria,* perfect balance of the ocular muscles. *Heterophoria,* imperfect balance of

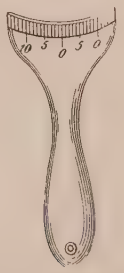

Fig. 56.—Strabometer. A simple but inaccurate instrument for measuring the amount of deviation in strabismus.

the muscles or latent squint. *Esophoria,* a tendency inward or latent convergence. *Exophoria,* a tendency outward or latent divergence. *Hyperphoria,* a tendency of the visual axis of one eye higher than the other.

Jackson applies the term *insufficiency of the ocular muscles* to latent paralytic squint and limits Stevens nomenclature to latent concomitant squint.

Cause.—The same as in manifest paralytic strabismus and manifest concomitant strabismus.

Diagnosis.—The *cover test* will reveal a latent squint of 2 or 3 degrees or more. It is made as follows: Have the patient fix one eye on an object at a distance of 20 feet.

Cover the other with a card. Binocular vision as well as diplopia are now impossible and therefore the influence which stimulates the patient to overcome this tendency toward deviation is no longer active. The muscle or muscles which have received the excess of innervation will relax and the covered eye will slowly assume the position in which the muscles are at rest. When the card is removed the covered eye will quickly return to the position of binocular fixation. The direction of this quick movement of the eye is opposite to the deviation and the amount of the deviation and recovery are equal.

There are numerous instruments, found in every eye clinic, for the determination of the nature and amount of latent squint. It is not necessary therefore to describe them in this abbreviated text.

Treatment.—The treatment of this condition is difficult and belongs to the oculist. Careful correction of any refractive error is of first importance. The constitution should be appropriately treated and use of the eyes regulated. In some cases wearing prisms gives relief. There are methods of exercising the weak muscle which may restore the necessary power. As a last resort operative measures may be adopted, which consist in partial tenotomy of the over-active muscle or advancement of the weak one.

NYSTAGMUS.

This condition is characterized by rapid, involuntary oscillation of the eyeballs, generally in the lateral direction. It may be congenital or acquired and nearly always affects both eyes. If acquired, the patient will, at first, complain of the movement of objects looked at.

Cause.—Defective development of the eyes, albinism, bad vision from corneal and lenticular opacities, blindness and protracted use of the eyes in an abnormal position, it being common with miners, who work with their eyes directed

obliquely upward. It is also due to brain lesions of central origin, ataxia and tumors of the cerebellum.

Treatment.—Improve vision by all possible means; if there is any refractive error put on the correcting glass; if there is a central corneal scar make a false pupil. In case of strabismus do a tenotomy, and if the occupation is at fault change it at once. The great majority of cases of nystagmus get little or no relief.

LESSON X.

DISEASES OF THE LIDS.

BLEPHARITIS.

This is an inflammation of the lid border characterized by the following symptoms given in the order of their severity. 1. Hyperemia, itching and slight swelling. 2. Seborrhea or hypersecretion of the sebaceous glands. The dried sebum forms yellow crusts on the border of the lid. 3. Ulceration at the root of the lashes. 4. Thickening of the edge of the lid. 5. Falling of eyelashes with atrophy of their follicles. 6. Ectropion with eversion of the lacrymal puncta and resulting epiphora.

FIG. 57.—Blepharitis, eyelashes matted into bundles by the secretion along lid borders.

Cause.—It is sometimes eczematous in nature and is most frequently found in the fair-skinned, the strumous and the badly nourished. Chronic conjunctivitis, lacrymal obstruction, errors of refraction, exposure and abuse of eyes are causative. It frequently follows the exanthematous fevers. The disease is limited to no age but is most frequent in children.

Treatment.— Correct the refractive error. Protect eyes from the irritation of dust, smoke, etc. Treat the constitution with cod liver oil, iron and arsenic if struma or debility are present. If there are ulcers around the cilia pull out the

lashes so affected and touch the ulcers with nitrate of silver stick. Rub into the edge of lids, once a day, an ointment of the yellow oxid of mercury, gr. ½ to vaseline 3i, or an ointment of ammoniated mercury, gr. ½ to 3i. Before applying the ointment all secretion should be cleansed from the lid border. Eight grains of biborate of soda to one ounce of warm water will be found useful in removing the

Fig. 58.—Chalazion of upper lid.

crusts. If there is lacrymal obstruction it must receive appropriate attention. In the chronic stage of blepharitis stimulating tar ointments are recommended but in the majority of cases mild and soothing measures will be the most efficacious.

HORDEOLUM.

A stye is an acute inflammation of a sebaceous gland at the lid border. It is usually, in appearance and symptoms, a small boil but sometimes produces general edema of the lid with chemosis of the conjunctiva.

Cause.—Error of refraction, general debility, constipation, possibly germ infection.

Treatment.—Use hot applications to bring the inflammation to a focus, then open. Correct the constitutional condition if debility exists. Correct refractive errors and remove any source of local irritation. Sulfid of calcium, ½ grain twice a day or dilute sulfuric acid ten drops after each meal may be given. A stye can sometimes be aborted in the earliest stages by the use of cold applications.

CHALAZION.

This small tumor of the lid is due to a chronic inflammation of a Meibomian gland. Hala claims this inflamma-

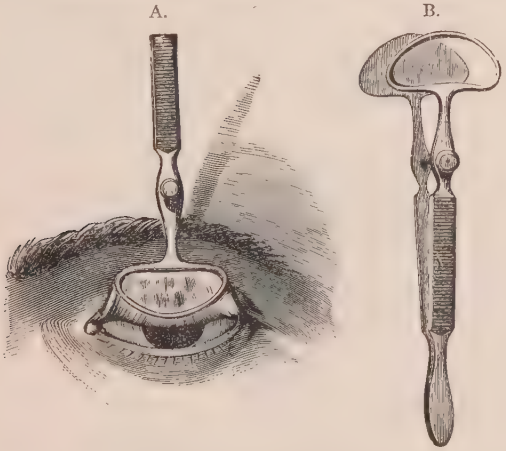

FIG. 59.—A, method of applying lid forceps for removal of chalazion through the conjunctiva; B, lid forceps.

tion to be the product of the xerosis bacillus. Its development is gradual and usually without any symptoms which annoy the patient. The inflammatory process causes proliferation of the epithelial lining of the gland and cell infiltration of the surrounding tissue. This inflamed area develops into a granulation mass surrounded by a thin connective tissue capsule. The granuloma tends to break down in the

center, forming a liquid, which may become purulent. Rarely the mass becomes fibrous and solid.

Treatment.—The contents may escape and the tumor disappear spontaneously. Sometimes they can be cured by hot applications, massage and ammoniated mercury ointment. An operation is generally necessary. The incision may be made through the skin or conjunctiva, depending upon the proximity of the tumor to these surfaces. If the incision is in the skin it should be made parallel with the lid border so as not to divide unnecessarily the fibers of the orbicularis muscle. If the incision is made through the conjunctiva it should be vertical to the lid border to avoid cutting the ducts of the Meibomian glands. If the tumor is soft a curette will remove it; if hard and fibrous it will have to be dissected out.

FIG. 60.—Trichiasis of upper lid.

TRICHIASIS.

Wild hairs, misplaced or misdirected eyelashes rubbing the globe, produce great pain and blepharospasm and may cause ulceration and subsequent opacity of the cornea.

Cause.—If the trichiasis is partial it may be congenital or may be due to the cicatricial contraction following styes, blepharitis ulcerosa, traumatism, etc. If there is a complete trichiasis it is usually associated with entropion and is, as a rule, the result of trachoma.

Treatment.—1. Epilation or pulling out of the offending hairs is only of temporary benefit, as they grow in again. 2. Electrolysis, introduced by Michel of St. Louis, is valuable. A needle attached to the negative pole is passed to the hair bulb, which is killed by a current of about five milliamperes. This procedure is exceedingly painful. 3. Excision of the misplaced hairs with their bulbs is useful when they are few in number and close together. 4. If the wild hairs are isolated their direction can be changed by passing a needle, threaded with a loop, through the lid in the direction you wish the hair to take, then catching the hair in the loop and drawing it through the tissues as you pull the thread through. 5. When the trichiasis is total and the lid border is turned inward, one of the operations for entropion should be done.

FIG. 61.—Entropion of the lower lid. (After Mackenzie.)

ENTROPION AND ECTROPION.

Entropion is a turning in of the lid. Ectropion is a turning out of the lid.

1. We have spasmodic entropion and spasmodic ectropion.

Spasmodic contraction of the fibers of the orbicularis near the lid border in conjunction with a relaxed and flabby skin and a deep set eyeball causes the lid border to turn inward, producing spasmodic entropion, which is nearly always found in the aged. This condition occurs almost without

exception in the lower lid. Spasmodic contraction of the fibers of the orbicularis farthest from the lid border, in conjunction with a tense skin, congested and thickened conjunctiva or a prominent eyeball, causes the lid border to turn outward producing spasmodic ectropion, which is nearly always found in children and young people.

2. We have cicatricial entropion and cicatricial ectropion.

Trachoma, some forms of conjunctivitis and wounds may produce cicatricial contraction of the conjunctiva lining the lid which turns the edge of the lid inward, causing entropion.

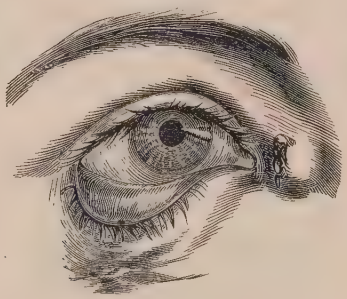

FIG. 62.—Ectropion of lower lid.

Burns, wounds, ulcers, caries of the orbital border and other causes may produce cicatricial contraction of the skin of the lid, which turns the edge of the lid outward, causing ectropion.

We have, in addition to the above conditions, a paralytic ectropion due to paralysis of the orbicularis muscle. The lower lid falls outward and away from the globe by its own weight. The lower lid is the only one affected by paralytic ectropion.

The treatment is almost always operative. Spasmodic entropion can be temporarily relieved by painting the skin over the inferior orbital margin with collodium, contraction of which everts the lid border.

ANKYLOBLEPHARON.

The edges of the upper and lower lid may grow together producing a condition called ankyloblepharon. It may be congenital or acquired; partial or complete. The cause is

Fig. 63.—Ankyloblepharon.

some accident or disease which leaves the lid margins in a denuded and granulating state. If in this condition the raw surfaces are kept in apposition they will grow together.

LESSON XI.

DISEASES OF THE LIDS (*Continued*).

BLEPHAROSPASM.

Spasm of the orbicularis appears under a variety of forms: 1. Abnormal frequency of winking or nictitation may be an unconscious habit which sometimes lasts a life-time. 2. A similar manifestation is seen in children, due to chronic conjunctivitis, but it may be the beginning of a general chorea. 3. In hysteria there is sometimes pronounced blepharospasm, which may be tonic or clonic. 4. In old age a tonic blepharospasm, which resists all treatment, may occur (Fuchs). 5. A reflex spasm of the orbicularis may be due to trichiasis, corneal and conjunctival diseases, foreign bodies, errors of refraction, and to any condition which can cause photophobia. Treatment is to remove the cause. 6. There is a clonic form of blepharospasm corresponding with tic douloureux, which is very painful. At given intervals the cramp seizes the orbicularis and other muscles of one side of the face, causing distortion and great pain. The paroxysm passes off in about a minute, to be repeated again after an interval varying in length in different cases. I have seen it return four or six times in an hour. The cause is some nerve or brain lesion, and treatment is very ineffectual. Iodid and bromid of potassium have each been beneficial. If any peripheral, exciting cause can be discovered, it should be removed.

LAGOPHTHALMIA.

Lagophthalmia is an inability to close the lids. Constant exposure of the globe causes conjunctivitis, ulceration of the cornea, and an overflow of tears, due to the mal-position of

the punctum. The evils of lagophthalmia are lessened by the tendency of the cornea to turn upward under the lid when an effort to close the palpebral fissure is made. This also occurs in sleep.

Cause.—1. Protrusion of the eyeball as in ex-ophthalmic goitre or orbital tumors. 2. Large anterior staphyloma. 3. Congenital shortening of the lids. 4. Loss of lid tissue from lupus, burns, etc. 5. Ectropion. 6. Paralysis of the seventh nerve. The course of this nerve is long and devious, and it passes through numerous tissues, which exposes it to accident or disease.

Treatment.—The treatment consists in removing the cause, meanwhile protecting the cornea from irritation by covering the eye with a bandage or holding the lids together with adhesive plaster. In the erect position, the force of gravity will help to draw the lid down over the cornea. In some cases tarsorrhaphy is necessary. This consists in shortening the palpebral fissure by uniting the edges of the lids.

PTOSIS.

Ptosis is a complete or partial drooping of the upper lid. Vision may be obstructed by the lids covering the pupils. To prevent this the patient throws his head backward and tries to raise the lids by elevating the brows. If congenital it is frequently bilateral; when acquired it is generally unilateral.

Cause.—The causes of congenital ptosis are: 1. Deficient development or absence of the levator palpebrae superioris muscle. 2. Injury inflicted by the forceps in difficult delivery. 3. Defective attachment of the skin to the underlying tissues, producing that form called ptosis adiposa, in which the skin falls over the lid border like a pouch.

The causes of acquired ptosis are: 1. Injury to the levator muscle. 2. Paralysis of the third nerve, usually from

syphilis (Fig. 64). 3. Thickening of the lids by new growths, trachoma, etc. 4. Hysteria.

Treatment.—Attack the cause when it can be located. The congenital forms require operations. In paralysis use anti-

Fig. 64.—Acquired ptosis of syphilitic origin. The effort to raise the lids by elevating the eyebrows is shown.

syphilitic and anti-rheumatic measures. Electricity, one pole back of the ear and the other over the lid, may be tried.

ECZEMA.

Eczema of the lids is most frequently met with in children who have phlyctenular ophthalmia, and in adults with an irritating discharge from the eye. The symptoms and treatment are the same as of eczema in other parts. In an acute case treatment should be mild and soothing; when chronic

it can be more stimulating. Dust with starch powder or aristol. Apply oxid of zinc ointment to which carbolic acid, 5 grains to the ounce, may be added. Yellow oxid of mercury ointment is useful, as is also painting with nitrate of silver solution, 10 to 20 grains to the ounce.

HERPES ZOSTER OPHTHALMICUS.

This term is applied to shingles following the course of the first and second divisions of the fifth nerve. It is characterized by redness and swelling of the skin and the formation of vesicles on the forehead, eyelids and nose. The disease is very painful and is a menace to sight if the vesicu-

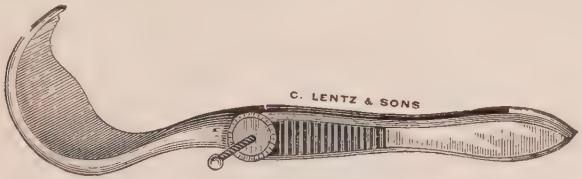

FIG. 65.—Clamp used to prevent hemorrhage and steady the part in lid operations.

lar eruption appears on the cornea. A severe neuralgia generally precedes the attack and may persist for a long time after it. The cause of the disease is an inflammation of the fifth nerve of an obscure character. Treatment is unsatisfactory. The vesicles should not be ruptured and when they dry forming crusts, the latter should remain undisturbed. Picking off the crusts deepens the subsequent scars. Anodynes may be required. Internally salicylic acid and quinin have both been recommended.

PHTHIRIASIS.

Crab lice may get into the eyelashes and give rise to excessive itching; the consequent rubbing and scratching of the lids sets up a mild inflammation which may be mistaken for blepharitis. The lice and their eggs may be seen on the cilia. Treatment is to rub the lid border and lashes thoroughly

with mercurial ointment every night until the parasites are killed.

ECCHYMOSIS OF THE LIDS.

A "black eye" is the result of any cause which ruptures a blood vessel of the subcutaneous tissue of the lid. It is most frequently due to a blow. Time is the only cure. A bandage, cold applications immediately after the accident, arnica or lead and opium wash will assist. When all subcutaneous oozing has ceased and the clot is formed its absorption may be hastened by hot applications and gentle massage.

Epithelioma or rodent ulcer of the lids is not a rare disease in elderly people. Its site of election is at the margin near the inner canthus and usually on the lower lid. It develops slowly. The absence of other evidences of syphilis will assist in differentiating it from a *syphilitic ulcer.* It can be distinguished from *lupus* by the fact that lupus begins much earlier in life and extends to the lids from its starting point on some other part of the face. Excision, caustics and cautery are the methods advised for its removal. The tendency to recurrence at times renders all these methods useless. The X-ray has been found curative in these cases and should be tried.

Sarcoma of the lids is rare. Lupus, molluscum contagiosum, xanthelasma, milium and nevus are diseases of the skin of the lids whose description belongs more properly to a work on skin diseases. Erysipelas may attack the lids, and if it extends to the orbital tissues there is some danger of serious consequences, such as orbital abscess, optic neuritis, optic atrophy and thrombosis of the retinal vessels.

The primary syphilitic sore has been known to appear upon the lids. The various eruptions of secondary syphilis and tertiary ulceration are more common. Syphilitic ulceration must not be mistaken for lupus or epithelioma.

LESSON XII.

DISEASES OF THE LACRYMAL APPARATUS.

EPIPHORA.

The lacrymal system is divided into a *secretory* part, the glands, and an *excretory* or drainage part, the puncta, canaliculi, sac and duct. Normally the lacrymal secretion is about balanced by evaporation. When the tears overflow on to the cheek the condition is called *epiphora.* It is the most con-

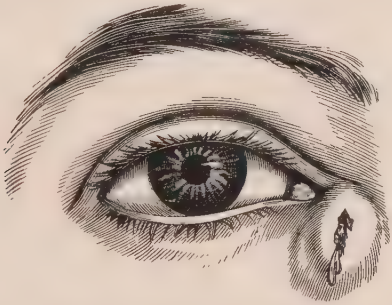

FIG. 66.—Fistula of lacrymal sac.

stant and significant symptom of disease of the lacrymal apparatus. There are two forms of epiphora; one related to the secretion and the other to the drainage of tears.

1. Epiphora caused by excessive secretion may be due to the influence of certain emotions such as grief or laughter; acute disease particularly of the cornea and iris; chronic conjunctivitis and irritation of the conjunctiva by foreign bodies, wind, dust or smoke; irritation of the retina by bright light; irritation or disease of the mucous membrane of the nose; and neuralgia of the fifth nerve. This form is called reflex epiphora.

2. Epiphora caused by impairment of the drainage apparatus may be due to eversion or occlusion of the puncta, plugging or stricture of the canaliculi, inflammation of the sac or stricture of the duct. The amount of epiphora resulting from defective drainage alone is either not appreciable or very slight as evaporation will about dispose of the normal secretion. It would follow then that obstruction of the drainage apparatus must be associated with some condition productive of hypersecretion of tears in order to cause epiphora. A moderate hypersecretion of tears will not cause epiphora if the drainage apparatus is patulous as the fluid will be carried into the nose, but with the drainage obstructed the smallest excess of tears must overflow the lids.

DISEASES OF THE LACRYMAL GLAND.

1. **Inflammation** of the lacrymal gland occurs very rarely. It may be acute or chronic. There would be the usual symptoms of inflammation which might result in suppuration or recover without it. There is often difficulty in excluding orbital cellulitis, phlegmon of the lid and periostitis, owing to the pronounced chemosis of the conjunctiva and great swelling and tenderness of the parts. Treatment consists of hot bichlorid fomentation, anodynes and evacuation of the pus if formed.

2. **Dislocation** of the lacrymal gland appears as a movable tumor under the ocular conjunctiva at the upper and outer part of the globe. Treatment does not avail. Extirpation may be resorted to.

3. **Tumors** of numerous varieties may develop in the lacrymal gland. Hypertrophy and atrophy have been observed. A tumor or hypertrophy of the gland would tend to force the eye ball downward and inward, causing diplopia. If the growth developed behind the ball exophthalmos would follow. Extirpation of the diseased gland is the only measure likely to prove beneficial.

4. **Fistula** of the gland is generally the result of an abscess or injury. A connection with the conjunctival sac should be established, then the cutaneous opening is easily closed by cauterization.

5. **Dacryops** is the term applied to a bluish, translucent, soft tumor which appears in the upper and outer conjunctival fornix. It is caused by the occlusion of one or more of the ducts which convey the lacrymal fluid from the gland into the conjunctival sac. As the tumor is a distended duct filled with tears it will collapse if punctured and the treatment consists in establishing a permanent opening.

ANOMALIES OF THE PUNCTA AND CANALICULI.

1. **Eversion,** or falling of the lower punctum away from the eyeball, may be due to ectropion, chronic conjunctivitis, blepharitis marginalis, lagophthalmia, or the relaxation of

FIG. 67.—Weber's straight canaliculus knife.

the lower lid found in old age. Though the upper punctum may be in its normal position, epiphora will follow any hypersecretion of tears.

2. **Obliteration** of a punctum or canaliculus may be congenital or may result from traumatism or chronic inflammation.

3 **Obstruction** of a canaliculus by a foreign body sometimes occurs.

CHRONIC DACRYOCYSTITIS.

This is a catarrhal inflammation of the sac and duct. *Stricture of the duct* will also be included under this head as these conditions merge into each other and are more or less interdependent.

Symptoms.—A slight catarrhal inflammation of the mucous membrane of the sac and duct, creates a muco-purulent

discharge, some of which passes backward through the puncta and produces a mild conjunctivitis and epiphora. This slight attack may disappear without treatment, or upon the instillation of some mild antiseptic collyrium and the appropriate attention to the nose. If the inflammation is more severe, the swelling of the mucous membrane will pro-

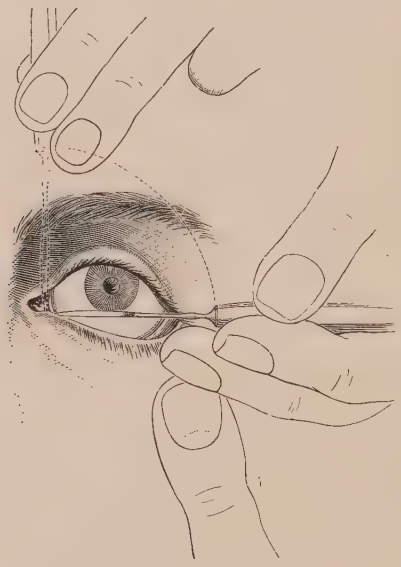

Fig. 68.—Slitting the canaliculus.

duce an occlusion of the duct and a consequent accumulation of the contents of the sac. The muco-purulent contents will become purulent, and will escape through the puncta and excite a conjunctivitis. With this conjunctivitis there will be hypersecretion of tears and epiphora. The accumulation of fluid in the sac produces a tumefaction which will disappear upon pressure, as the fluid is forced back through the puncta or through the stricture into the nose. The stenosis of the duct may be complete. The purulent contents of the

distended sac are extremely toxic and will almost surely infect a wound of the cornea, will often light up an active inflammation of the connective tissue surrounding the sac (acute dacryocystitis), and may, if of long standing, produce caries of adjacent bone.

Cause.—Dacryocystitis may be started by any of the numerous causes of inflammation of mucous membrane, such as temperature changes and infection. Stricture of the duct will cause a dacryocystitis, and stricture may be due to morbid conditions of the nasal cavities, traumatism, asymetry of the face, deflected septum, periostitis or syphilis. The prog-

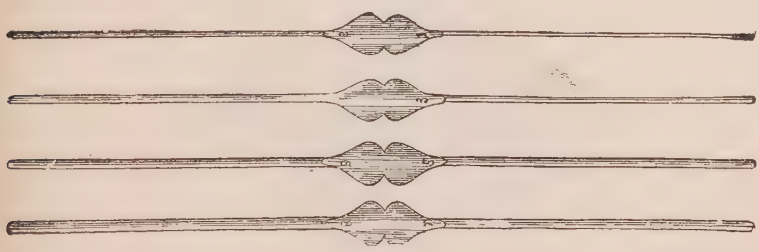

FIG. 69.—Bowman's probes for dilating the nasal duct.

nosis in chronic cases is bad. If cured they require months of treatment, and too often patients have not time or inclination to resort to the needed measures.

Treatment.—Teach patients to keep the sac empty by pressure. See that the nasal cavities are kept clean by washing them out with Dobell's solution. The mildest form is sometimes benefited by dropping into the eye, three times a day, a 1 to 2,000 solution of blue pyoktanin, or a weak sulfate of zinc or alum solution. Wash out the sac with a 1 to 10,000 solution of bichlorid of mercury, or a warm boracic acid solution, every other day. If the discharge is purulent, inject into the sac a small quantity of a solution of nitrate of silver (gr. 2 to the ounce), protargol (5 to 20 per cent.), or argyrol (10

per cent.), after having cleaned it out by washing with bichlorid of mercury or boracic acid solutions. If there be a stricture of the duct slit a canaliculus (upper preferred) and dilate the stricture with probes.

These two procedures, though simple, would better be learned clinically. Great care must be observed in probing,

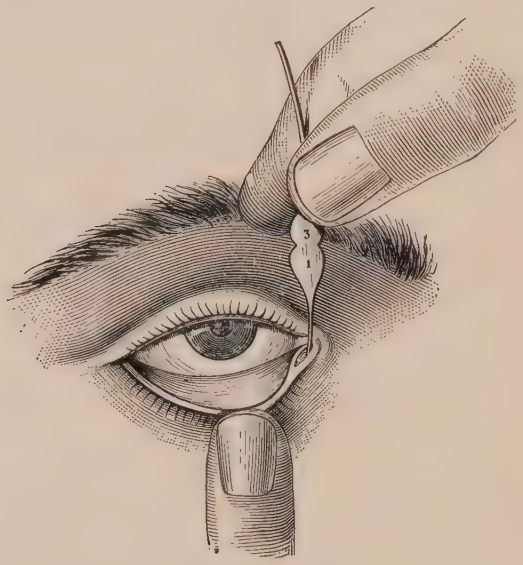

Fig. 70.—Probing the nasal duct.

as there is danger of lacerating the tissues around the sac. This is particularly true of the early stages of treatment when small probes are being used. A preliminary injection of cocain into the sac will render the operation less painful. The use of extract of the supra-renal gland, by reducing the vascularity of the mucous membrane of the duct seems to facilitate the passage of probes. When the probe is removed the sac and duct should be treated with an injection of one of the antiseptics or silver preparations above mentioned.

How often to probe, how long to leave the probe in the duct, and the maximum size of the probe to be used are questions upon which various opinions are entertained.

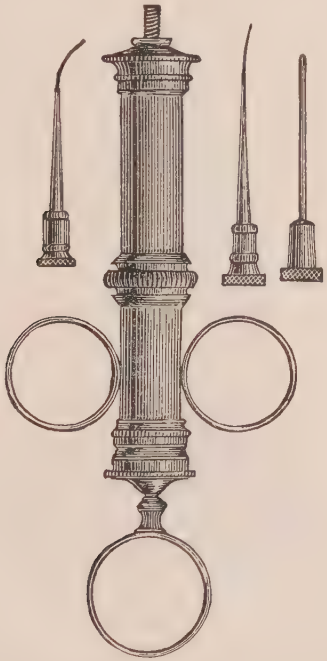

Fig. 71.—Syringe for injecting the lacrymal passages.

ACUTE DACRYOCYSTITIS.

Symptoms.—In the course of a chronic dacryocystitis, a severe inflammation may suddenly develop in the region of the sac. There will be redness and swelling which will extend to the lids and conjunctiva. Pain will be very severe and there may be some fever. Pus will form and the skin over the abscess become thin. Unless opened the skin will break, emptying the contents of the abscess and establishing

a *lacrymal fistula* (Fig. 66). As soon as the pus is evacuated the symptoms rapidly subside, to recur as soon as the fistula is allowed to close.

Cause.—A lesion of the mucous membrane of a sac affected by chronic dacryocystitis, allows its toxic contents to infect the surrounding sub-mucous tissue and the active phlegmonous inflammation follows.

Fig. 72.—Acute dacryocystitis.

Treatment.—Evacuate the pus by slitting up a canaliculus if possible, if not open through the skin over the sac. Cleanse with some antiseptic solution and if the swelling and pain are considerable use hot, antiseptic compresses. Keep the incision open by gauze drainage until the abscess can be cleansed through a canaliculus and then treat as a chronic dacryocystitis.

In obstinate cases of dacryocystitis it is sometimes deemed expedient to completely obliterate the drainage passages. For example, a cataract operation should never be attempted

in the presence of a discharge from the lacrymal sac, owing to the imminent danger of infecting the corneal wound. The sac may have to be obliterated to stop the discharge. This is done by dissecting it out or destroying it with the actual or chemical cautery.

LESSON XIII.

DISEASES OF THE ORBITS.

MENINGOCELE.

Sometimes there is defective development of the bones of the orbital rim where the nasal wall and roof of the orbit unite. The result is a congenital, cystic tumor in this region, composed of the meninges of the brain, filled with cerebral fluid. The tumor is called a *meningocele,* or if it contain brain substance, an *encephalocele.* It may be mistaken for an ethmoidal mucocele, sebaceous cyst or dermoid tumor. The following diagnostic points should be remembered:

1. It is congenital.
2. It gives the impression of being firmly attached to the bone.
3. There is a pulsation of the cerebral fluid in a meningocele synchronous with the heart beat.
4. Steady pressure will force some of the contents of a meningocele back into the cerebral cavity and then the opening in the bony wall may be felt with the point of the finger.
5. Symptoms of increased cerebral pressure may be excited when the fluid is forced back into the cerebrum.

A meningocele should not be disturbed.

PERIOSTITIS.

Periostitis may attack any part of the orbit but is most frequent at the margin. When superficial the hard swelling and tenderness upon pressure make the diagnosis simpler than when deep in the orbit. The latter form is often hard to differentiate from orbital cellulitis until a fistula is formed and rough bone can be detected with a probe.

Symptoms.—Before entering upon the symptomatology of the few inflammatory conditions to which the orbit is subject, it is well to mention that there are a number of symptoms common to nearly all diseases of the orbits. Of these there are two so constantly in attendance as to make them worthy of special mention.

1. Proptosis or exophthalmos.
2. Limitation of movement of the eyeball.

Associated with these salient symptoms are diplopia, injection and chemosis of the conjunctiva, redness, swelling and edema of the lids and severe pain, most noticeable when the patient attempts to move the globe, or the surgeon presses it backward into the orbit.

The character of the symptoms in periostitis will depend upon whether the inflammation is checked in the stage of periosteal thickening or goes on to suppuration. Also whether acute or chronic, circumscribed or diffuse, external or deep seated.

When external all the signs of local inflammation will be present. Unless checked in the first stages, pus will form under the skin and when discharged a sinus will be established through which rough bone can be detected. The discharge keeps up for a variable period and when the fistula is healed it leaves the characteristic funnel-shaped dimple in the skin, which is adherent to the underlying bone. If there is much contraction of the skin ectropion results.

When deep-seated and non-suppurative the symptoms will be pain and probably protrusion of the ball with some limitation of its movement. When suppuration takes place the symptoms are practically those of orbital cellulitis, page 89. Caries and necrosis may result. Periostitis of the roof of the orbit is the kind most likely to cause meningitis, owing to the thinness of the bone here and the proximity of the brain.

Cause.—Injury, syphilis, scrofula and rheumatism. When syphilitic it is usually a tertiary manifestation.

Treatment.—The constitutional treatment appropriate to the cause should be instituted. Locally apply hot compresses, evacuate pus when formed, establish drainage and wash out antiseptically. Correction of any resulting deformity such as ectropion or lagophthalmia should not be attempted until the periostitis is entirely well.

CARIES AND NECROSIS.

Caries and necrosis occur most frequently at the margin of the orbit, owing to its greater exposure to injury. They generally begin as a periostitis. The course of the disease is chronic, sometimes covering a period of several years. A bad scar is the usual result, the skin contracting and adhering firmly to the underlying bone. The danger of meningitis is greatest when the horizontal plate of the frontal bone is the part involved. The treatment consists in establishing good drainage and cleansing frequently with antiseptic injections.

HYPEROSTOSIS, PERIOSTOSIS AND EXOSTOSIS.

Hyperostosis (thickening of bone), periostosis (thickening of the periosteum), and exostosis (new bone developed from the periosteum), may occur in the orbit. The symptoms will depend upon the extent and location of the process. If of any size exophthalmia will be noticed. If located near the sinuses at the apex, the circulation in the orbit may be interfered with or pressure may be exercised upon the optic nerve or nerves supplying the ocular muscles. The treatment consists of alteratives internally or surgical removal.

INJURIES OF THE ORBIT.

Fractures may be marginal or deep seated. Deep seated fractures may be the result of force applied directly or indirectly. There will be hemorrhage into the orbit with protrusion of the ball. If the inner wall is fractured blood

will escape through the nose and air may be forced into the cellular tissue of the orbit and lids producing *emphysema.* Direct fractures of the roof, for example, from a knife or cane are exceedingly dangerous to life, owing to the injury to the brain which is almost inevitable. Fractures extending into the optic foramen are dangerous to vision, owing to direct injury to the nerve or hemorrhage into its sheath.

The contents of the orbit are subject to all kinds of injuries. Many remarkable cases have been reported, showing the great tolerance of the orbital tissues to the presence of foreign bodies. The following, reported by Carter, will suffice. A man fell down a flight of steps, at the bottom of which was a row of hat pegs. He cut his eye lid, but did not consult a surgeon for a few days. The surgeon had treated him several days before noticing a foreign body in the wound. It proved to be the shaft of a hat peg 3¼ inches long. The X-ray is of great value in determining the presence and location of foreign bodies in the orbit.

The treatment of all orbital injuries should be based upon established surgical principles. In the use of antiseptics, however, the sensitiveness and delicacy of the conjunctiva and cornea must not be overlooked.

ORBITAL CELLULITIS.

This is an inflammation of the cellular tissue of the orbit which may be acute or chronic, but usually results in suppuration with the formation of an abscess.

Symptoms.—Proptosis with diplopia, pain, limitation of movement of the ball, injection and chemosis of the conjunctiva and swelling and redness of the lids. As the severity of this disease varies greatly in different cases, we shall expect variation in the degree of manifestations of all symptoms. In the severe forms there will be chills with fever, and may be loss of vision due to pressure upon the optic nerve or disturbance of the intra-ocular circulation. There may be

ulceration of the cornea and possibly suppuration of the whole eyeball. The inflammation has been known to extend to the meninges of the brain.

Cause.—The causes are such as produce cellulitis in other locations and are numerous. They may be traumatic or idiopathic. Special mention may be made of the severe form due to erysipelas, and also to the fact that it may arise by metastasis in all pyemic conditions, or puerperal septicemia.

FIG. 73.—Double orbital cellulitis, the result of erysipelas. (De Schweinitz.)

It may follow thrombosis of the ophthalmic vein and has been known to result from abscesses of the teeth in the upper jaw. Suppuration in the adjoining cavities (frontal sinus, ethmoid cells and sphenoidal sinus), may extend to the orbit.

Treatment.—Support with tonics, especially quinin and iron. Relieve pain by anodynes. Apply hot fomentation, and as soon as the abscess can be located or any sign of fluctuation appears, open and treat antiseptically. When the exophthalmos is very pronounced and the pain intense it is advised to make an incision into the orbit, through the con-

junctiva without waiting for evidences of suppuration. In making the incision the flat side of the knife should be directed toward the ball and the ocular muscles avoided.

Inflammation of the capsule of Tenon has been known to occur without involving the cellular tissues of the orbit. When idiopathic it is generally rheumatic in origin and the treatment should be directed to that dyscrasia. The traumatic form has often been caused by infection following an operation upon the muscles for the correction of strabismus. The treatment is the same as in orbital cellulitis.

TUMORS OF THE ORBIT.

The orbit contains many different tissues, consequently a great variety of tumors may develop in this locality. Those which originate in adjoining cavities may reach a large size before any symptoms of orbital disease become manifest. It is therefore well to determine, if possible, whether other cavities are involved. When there is an exophthalmos, which has developed slowly, combined with limitation of movement, without the usual manifestations of inflammation, the diagnosis of a tumor may be arrived at with a degree of assurance. If, however, the tumor has developed rapidly and is associated with symptoms of active inflammation, the differentiation from periostitis, orbital cellulitis, etc., may be difficult.

If the nature and extent of the growth will admit, it should be removed without the ball. To accomplish this it may be necessary to temporarily resect a wedge-shaped piece of the outer orbital wall. When the tumor is malignant the most radical removal of eye ball and orbital contents secures no immunity from a probable recurrence.

PULSATING EXOPHTHALMOS.

This condition may develop slowly but its onset is usually sudden. The patient may be conscious of a rupture or giving way of some structure in the head. This sensation is soon

followed by protrusion of the ball, congestion of the conjunctiva, swelling of the lid, pain, pulsation of the eye ball, and a bruit heard over the orbital region. If firm pressure is applied over the eye it can be forced back into its normal position. When back the bruit may no longer be heard and the roaring sound, so annoying to the patient, generally ceases. Sometimes vision is much impaired and the ophthalmoscope shows a swelling of the optic disc with distortion and enlargement of the retinal veins.

Cause.—It is generally due to rupture of the internal carotid artery within the cavernous sinus. It may be traumatic or spontaneous. If the latter there must have been a diseased condition of the artery which thinned its walls. Aneurism of the ophthalmic artery is sometimes the cause.

Treatment.—The object of treatment is to establish a clot in the ruptured artery. Compression of the common carotid should be tried. Digital or instrumental pressure should be exerted for as much of the time of each day as the patient can endure it. If this fail, ligation of the common carotid must be resorted to. If the patient escapes the dangers incident to this operation the pulsating exophthalmos will usually be found cured.

LESSON XIV.

DISEASES OF THE CONJUNCTIVA.

CATARRHAL CONJUNCTIVITIS.

This is the most frequent disease of the eye. It usually attacks both eyes, varies greatly in severity and duration, and tends to spontaneous recovery, rarely lasting over two weeks. *Hyperæmia* of the conjunctiva is generally given as

Fig. 74.—Conjunctival congestion. The circumcorneal zone the last part to become injected.

a separate disease but practically differs from simple catarrhal conjunctivitis only in degree, being milder.

Symptoms.—1. **Congestion** of the palpebral and ocular conjunctiva, the pericorneal zone remaining normal or the last part to become red. (Fig. 74.)

2. **Pain** of a scratchy, burning kind, feeling often as if there was a foreign body under the lids.

3. **Vision** slightly diminished owing to the presence of mucus and pus on the cornea.

4. **Discharge** of a muco-purulent nature which mats the lashes into small bundles and sticks the lids together during sleep.

5. **Photophobia** or intolerance of light.

6. **Swelling** of the lids (slight) and some thickening of the conjunctiva.

Cause.—Foul atmosphere, dust, smoke, wind, heat, cold, the glare of the sun, and errors of refraction. The exanthematous fevers, diseases of the lacrymal sac and duct, nasal catarrh and hay fever. A very contagious form of catarrhal conjunctivitis, which at times becomes epidemic, is caused by a small bacillus described by Weeks, and a conjunctivitis clinically very similar to that produced by the Weeks bacillus is due to the pneumococcus. Also a mild but persistent form of catarrhal conjunctivitis is associated with the presence of the diplo-bacillus of Morax and Axenfeld.

Treatment.—Remove the cause if discovered. Rest eyes and keep them clean. Use a cold compress as follows:

℞

Acidi Borici	ʒi
Tincturae Opii deodoratae	ʒvi
Aquae destellatae, q. s. ft.	℥viii

This is to be applied to the outside of the closed lids, on a thin cloth, folded once or twice, for fifteen minutes at a time, four times a day. The solution should be ice cold when used and the wet cloths changed every minute. Apply a weak yellow oxid of mercury ointment or boric acid salve to the edge of the lids at night to prevent adhesion. If discharge is profuse or purulent, paint everted lids, once a day, with a

solution of nitrate of silver, five to ten grains to the ounce, or a 2 to 5 per cent. solution of protargol. One drop of a 10 per cent. solution of argyrol, three times a day, is frequently beneficial. Argyrol is painless and practically non-irritating. A fresh solution should be used. Astringent collyria containing sulfate of zinc, tannin, alum, etc., are very popular. They are, in my estimation, not as useful as the above harmless application and are capable of mischief if, through an error of diagnosis, they are used in iritis, cyclitis or acute keratitis. An exception should be made of the diplo-bacillus conjunctivitis, in which form sulfate of zinc is particularly valuable.

CHRONIC CATARRHAL CONJUNCTIVITIS.

Symptoms.—After the subsidence of an acute attack the same general symptoms may persist in a milder form, or they may develop slowly without an acute manifestation. In the chronic form the palpebral conjunctiva and the fornix are the parts chiefly involved.

Cause.—The same agents which produce acute catarrhal conjunctivitis, but especially those which are slow and continuous in their action.

Treatment.—The source of any chronic irritation should be removed and the same line of treatment as recommended for an acute attack instituted. Stronger remedies are more applicable to the chronic form, and zinc, alum, argyrol, nitrate of silver, protargol or sulfate of copper may be used. Caution should be observed in the continuous use of the silver preparations owing to the danger of producing a dark, muddy discoloration of the conjunctiva called *argyria.*

PURULENT CONJUNCTIVITIS

This condition may be divided into two forms: 1. The infantile variety or *Ophthalmia Neonatorum,* which arises between the third and eighth day after birth and generally attacks both eyes. 2. The adult variety, or *Gonorrheal Ophthalmia,* which may attack but one eye.

Symptoms.—The period of incubation varies from 6 to 60 hours. The disease may be divided into three stages: Stage of infiltration, which lasts from 3 to 6 days; stage of pyorrhea, which lasts from 3 to 6 weeks, and stage of chronic blennorrhea, which varies greatly in duration.

1. **Congestion** of the palpebral and ocular conjunctiva.

2. **Pain** is severe and of a smarting, burning variety. The great thickness and weight of the lids causes also a continuous dull ache in the eye.

Fig. 75.—Purulent conjunctivitis.

3. **Discharge** is profuse and of a thin ichorous, beef juice kind in the first stage which changes in the second to thick yellow pus.

4. **Swelling** of the lids is so intense as to interfere with the proper inspection of the eye. When the pus begins to flow freely this swelling usually decreases. The conjunctiva becomes so edematous (chemosis) as to overlap the circumference of the cornea.

5. **Vision** may be interfered with by the pus on the cornea, by corneal ulceration, or by the inability to raise the upper lid.

6. **Ulceration of the cornea,** the result of disturbed nutrition and infection, may supervene. This is the most dan-

gerous symptom owing to the possibility of permanent scars, intra-ocular infection, and panophthalmitis.

Cause.—Inoculation with gonorrheal virus, the gonococcus of Neisser being found in the discharge. There are mild types which clinically simulate purulent conjunctivitis in which the gonococcus cannot be demonstrated. If the gonococcus is absent in the infantile variety, the disease has been caused by a vaginal discharge other than gonorrheal. Such cases are always mild. If the gonococcus is absent in the adult form, the disease must be due to other pus-producing germs. It will sometimes follow mechanical and chemical accidents or badly treated catarrhal conjunctivitis.

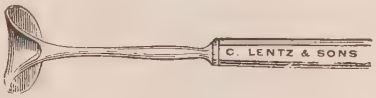

Fig. 76.—Desmarre's lid elevator.

Treatment.—In the stage of infiltration cold applications must be kept on the eye continuously. This may be done by keeping a number of small squares of muslin on a block of ice, and transferring one to the eye every minute. However, judgment must be exercised in the application of cold as it is a well known fact that the puny and scrofulous bear it badly. The secretion should be washed away with a warm boric acid solution (3 per cent.), or some other mild antiseptic, about once every hour or oftener. The bowels should be well purged with salines and the patient kept as quiet as possible. If pain is very severe an adult may be given an anodyne. In the stage of pyorrhea the mechanical cleansing of the conjunctiva must be vigorously continued. The upper lid should be turned once a day and its conjunctival surface painted with a 2 per cent. solution of nitrate of silver. In the place of nitrate of silver one of the less irritating silver preparations may be used. Protargol solution

(5 to 20 per cent.) or Argyrol solution (10 to 25 per cent.) may be dropped between the lids every three hours. Argyrol is practically non-irritating and is probably the best remedy we have. If the swelling of the lids is so great as to prevent eversion or to endanger the circulation, the outer canthus should be slit with a pair of scissors (canthotomy). In the second stage cold applications should be diminished, if not altogether discontinued, owing to their depressing influence upon the nutrition of the cornea. If the cornea be-

FIG. 77.—Application of Buller's shield. (De Schweinitz.)

comes hazy or shows a spot of ulceration hot applications should be applied, for 15 minutes at a time, 4 or 5 times a day; atropin or eserin dropped in the eye and the general treatment for corneal ulcers (page 122) followed as closely as is possible under the circumstances. In the third stage of the disease the treatment advised for chronic catarrhal conjunctivitis (page 95) should be followed.

In adults, where one eye is affected, protect the good eye by covering it with a watch crystal held in position by adhesive plaster. This is called Buller's shield. As the secretion is most liable to get into the good eye by flowing across

the root of the nose, the shield should be well sealed at this point by the use of cotton and collodium. To prevent ophthalmia neonatorum in a child born of a diseased mother, resort to the method of Credé, which is to wash its eyes thoroughly just after birth and drop between the lids several drops of a five grain to the ounce solution of nitrate of silver. Credé advised a ten grain to the ounce solution, but this has proved unnecessarily strong. There is every reason to believe that argyrol or protargol, in the proper strength, are as efficient prophylactics as the nitrate of silver.

There is a law in many States punishing midwives and nurses for not immediately reporting, to a health officer, the appearance of inflammation in the eyes of a new born under their care. A few convictions under this law would materially lessen the blindness from ophthalmia neonatorum.

MEMBRANOUS CONJUNCTIVITIS.

The characteristic feature of this inflammation is a plastic, fibrinous, pseudo-membrane on the tarsal and sometimes on the ocular conjunctiva. With the exception of this membrane the symptoms are very similar to those of purulent conjunctivitis. It is customary to divide this affection into *croupous* and *diphtheritic* conjunctivitis, but since the disease appears in every degree of severity, from an almost harmless condition to one of a most destructive character, it is difficult to draw a dividing line clinically. Usually the croupous form is a mild disease, rarely results in any permanent injury and runs a chronic course, while diphtheritic conjunctivitis is acute, severe and destructive. However nothing but the presence of the Klebs-Löffler bacillus will enable us to make a positive differentiation. The disease is rare.

Symptoms.—1. **Congestion** of the conjunctival vessels is hidden by the plastic membrane in severe cases. In a mild case, the plastic membrane being confined to the lids, the ocular conjunctiva will appear injected.

2. **Pain** is generally of an itching, burning character, but when there is great swelling of the lid there is an added sensation of pressure on the ball.

3. **Discharge** is at first serous and flaky, and may be tinged with a little blood. As soon as the membrane begins to soften the discharge becomes purulent.

4. **Swelling** of the lids is almost imperceptible in the mild forms but in a severe diphtheritic conjunctivitis the upper lid may become so thick and tense as to render its eversion impossible. The exudation into the conjunctiva may be so excessive as to shut off the circulation, producing gangrene and subsequent cicatricial contraction and adhesions.

5. **Vision** is affected as in purulent conjunctivitis (page 96).

6. **Ulceration of the cornea** is produced as in purulent conjunctivitis (page 96). In croupous conjunctivitis it rarely happens but in diphtheritic conjunctivitis, if severe, it is almost inevitable.

7. **The membrane** in mild cases is limited to the palpebral conjunctiva and can be wiped off leaving a slightly bleeding surface. In severe cases it covers the entire conjunctiva and can only be removed by force, leaving a raw surface.

8. **Constitutional Symptoms** will only be present when the disease is diphtheritic.

Cause.—When the Klebs-Löffler bacillus can be demonstrated the nature of the disease and its cause are definitely determined. The exact cause of those cases in which this bacillus cannot be demonstrated is, as yet, unknown.

Treatment.—For mild cases follow the treatment recommended for catarrhal conjunctivitis (page 94) being careful though not to use nitrate of silver until the membrane has disappeared. Before the separation of the membrane cleansing the conjunctival sac three or four times a day with an

antiseptic solution (bichlorid 1 to 5,000) is advised. In the severe form follow the treatment as suggested for purulent conjunctivitis (page 97) except that cold applications must not be used as continuously owing to greater danger of depressing the circulation, and nitrate of silver must be applied with caution and then not until the membrane has been thrown off. If the diphtheritic bacillus can be demonstrated constitutional treatment, including anti-toxin injections, should be instituted at once.

LESSON XV.

DISEASES OF THE CONJUNCTIVA (Continued).

GRANULAR CONJUNCTIVITIS OR TRACHOMA.

The characteristic feature of this disease is hypertrophy of the conjunctiva and the appearance in that membrane of small granular bodies. Trachoma may assume three forms:

1. **Papillary trachoma** in which the characteristic feature is hypertrophy of the conjunctiva. The normal papillae are greatly increased in size, hence the name. This form is also called chronic conjunctival blennorrhea, as there is always a variable amount of pus in the discharge. Notwithstanding the absence of the trachoma follicles the conjunctiva undergoes cicatricial changes and the sequellae are practically the same as when the granules are present.

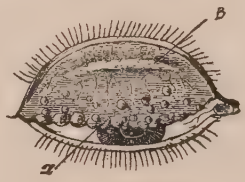

FIG. 78.—Granular upper lid. a, granulations; b, line of scar, in typical position parallel with border of lid. (Nettleship.)

2. **Granular trachoma** in which the characteristic feature is the appearance in the conjunctiva of small follicles or granules. These follicles are composed of lymphoid cells and connective tissue cells surrounded by an ill-defined fibrous capsule. They are imbedded in the fibrous layer and have a yellowish or grayish appearance. They develop later into connective tissue which undergoes cicatricial contraction. The follicles are most numerous in the fornix, but may be found in any part of the palpebral conjunctiva.

3. **Mixed trachoma,** which is the form under which we generally see the disease, is a combination of the two preceding varieties.

Symptoms.—The eyes are irritable, giving distress under exposure or misuse. The lids may be swollen and may droop a little. There is a slight muco-purulent or purulent discharge and there is a scratchy feeling under the lids. Upon inspection of the palpebral conjunctiva the characteristic appearance above described will be found. If the dis-

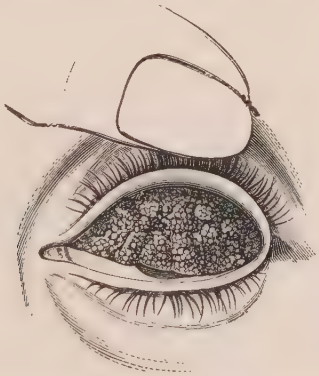

FIG. 79.—Exuberant granulations. No indications of cicatrization are present. (Jones.)

ease is of the papillary form the conjunctiva will have a rough or velvety appearance due to the enlarged papillae and the color of the swollen conjunctiva is of a slightly bluish tinge. There will also be some pus in the conjunctival fornix. If the disease is of the granular form the peculiar follicles will be present, but as stated above, the usual picture is a combination of these varieties. With the progress of the disease all the symptoms increase in severity. Cicatricial changes will take place in the conjunctiva and even in the underlying tarsus, rendering the mucous membrane hard and fibrous in parts and by its contraction bending the tarsus so as to pro-

duce trichiasis and its attendant evils. The ocular conjunctiva will become injected and *pannus* will develop (page 124). Ulceration of the cornea is a frequent complication and iritis may occur. Trachoma exhibits a marked tendency toward remissions and relapses. As a rule the disease covers a period of years unless persistently and successfully treated. Some cases seem incurable; they will relapse until vision is practically destroyed.

Cause.—Trachoma is generally conceded to be contagious, and the principle of the contagium is supposed to be a micro-organism. Numerous trachoma germs have been described, but none of them has as yet been accepted as the

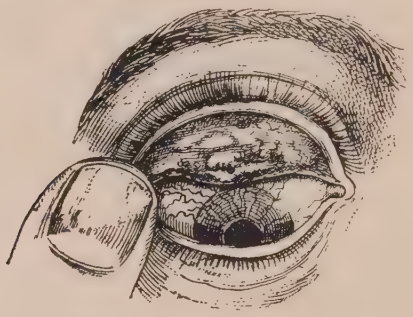

FIG. 80.—Trachoma and pannus. (Berry.)

specific cause of the disease. The fact that one eye may be affected for years without infecting its fellow is adduced as an argument against the contagious theory. The tendency of the disease to spread in crowded institutions is in favor of this theory. Certain races, among which are the Jews and the Irish, show a predisposition to trachoma, whereas the negro of our Southern States is almost immune.

Treatment.—Nitrate of silver, ten grains to the ounce, applied to the conjunctiva of the everted upper lid, once a day or every other day, depending upon the effect, is a val-

uable remedy. When nitrate of silver is applied to the conjunctiva a white coagulum is at once formed. The amount and rapidity of the formation of this coagulum indicates the activity of the remedy. When the effect desired has been gotten the action of the silver should be checked by a few drops of a solution of common salt. During the above application the cornea should be protected as much as possible. Bichlorid of mercury solution (1 to 5,000), Protargol (20 per cent.) and Argyrol (25 per cent.), are all useful agents. They should be applied directly to the conjunctiva of the upper lid. Rubbing them in to the membrane with more or less force, by means of a cotton applicator, has been recommended. These remedies are particularly applicable to the treatment of papillary trachoma. For cases in which the follicular feature predominates, the sulphate of copper, lapis divinus or alum stick is the best remedy. This should be applied lightly or thoroughly, daily or with longer intervals, depending upon the effect in each case. In mixed cases it is well to first reduce the papillary swelling with nitrate of silver, argyrol or bichloride of mercury and then treat the granular trachoma with bluestone. Cold applications are often soothing and beneficial. In the late cicatricial stages ointment of yellow oxid of mercury (grains iv to the ounce) or corrosive sublimate (gr. $\frac{1}{20}$ to the ounce) are recommended. Boroglycerid (30 per cent.) and glycerole of tannin (5 to 25 per cent.) may be tried.

Where the appearance of the granules indicates its feasibility, squeezing them out with Knapp's roller forceps facilitates the cure. This should be thoroughly done under an anesthetic, and the lids subsequently treated with argyrol or bichlorid of mercury solutions. Old cases, in which there is considerable pannus, as shown in Fig. 80, are often greatly improved by the use of an infusion of jequirity. The cases on which jequirity is used should be carefully selected and

as its use is sometimes attended with danger, this treatment should be left to an oculist. The use of jequirity or its substitute jequiritol will sometimes cause dacryocystitis.

ACUTE TRACHOMA.

During the course of a chronic trachoma the diseased eyes may take on a severe acute inflammation or the disease may seem to originate with an acute attack. Such an inflammation is spoken of as acute trachoma but is in reality a case of chronic trachoma plus an acute conjunctivitis.

Symptoms.—Rapid swelling of the lids and hypertrophy of the conjunctiva. Pain, which may extend to the brow and temples, lacrymation, heat, photophobia and congestion, with a muco-purulent discharge. The palpebral conjunctiva

FIG. 81.—Knapp's roller-forceps.

is swollen, red and shiny. The translucent granules, that are covered by the hypertrophied epithelium, usually are not seen until the acute symptoms subside. This occurs in from one to three weeks. It will often be impossible to distinguish this disease from acute catarrhal conjunctivitis until the granules appear.

Treatment.—Apply iced compresses or the cold boric acid and tincture of opium solution recommended on page 94. Ten per cent. argyrol solution may be of service. Distress will sometimes be so great as to warrant the use of bromids or morphin. When the swelling and pain have subsided and the granules appear, treat as a case of chronic trachoma.

FOLLICULAR CONJUNCTIVITIS.

This disease is sometimes described as a form of trachoma, as they are frequently almost identical in appearance. That there is a distinct difference is proven by the fact that

follicular conjunctivitis never permanently injures the conjunctiva, whereas trachoma always does.

Symptoms.—The symptoms are those of an acute or chronic catarrhal conjunctivitis to which is added the appearance of the follicles in the fornix of the lower lid, rarely in the upper lid. These granules, about the size of a pin head, are composed of adenoid tissue, identical with that of the true trachoma follicle. They may be few in number or very numerous; if the latter, they are usually arranged in longitudinal rows. The disease is most frequent in children and young people and is very prolonged and obstinate

Fig. 82.—Follicular conjunctivitis. (After Eble.)

in its course. At times it gives so little annoyance that its presence is discovered by accident.

Cause.—The etiology is obscure. It is supposed to be contagious as so many of the inmates of schools and institutions are attacked at the same time. Bad hygienic surroundings seem to be factors in the production of the disease.

Treatment.—The same treatment as advised for acute or chronic catarrhal conjunctivitis is applicable. An ointment of acetate of lead (gr. i to ʒi) is recommended but acetate of lead must never be used if there is any implication of the cornea. If the follicles are prominent, expression with the roller forceps will hasten the cure. Fresh air, good food, proper exercise, attention to refractive errors and the proper use of the eyes must not be overlooked.

LESSON XVI.

DISEASES OF THE CONJUNCTIVA (Continued).

VERNAL CONJUNCTIVITIS, OR SPRING CATARRH.

This is a disease of childhood or early youth. It affects both eyes and comes on with the advent of warm days (hence the name), and will frequently last until cold weather. There is a tendency to recurrence in the same individual year after year. The attacks, however, gradually cease and no injurious sequelae are left.

Symptoms.—The palpebral conjunctiva will be hyperemic and the swollen papillae assume a flattened, rectangular shape. Frequently it appears to be overlaid by a bluish white film as if covered by a thin layer of milk. The ocular conjunctiva is usually hyperemic and in typical cases presents a narrow band of grey, hypertrophied tissue at the limbus. This swollen mass may appear at the inner and outer side of the cornea or may entirely encircle it.

Frequently it forms an arch over the upper half of the cornea. The thickened tissue is sometimes interrupted at intervals by depressions which give it a nodular or bead-like appearance. It usually encroaches upon the cornea for a slight distance. The objective symptoms may be limited to the changes in the tarsal conjunctiva, or the circumcorneal hypertrophy may be the only objective manifestation but a typical case will present involvement of both.

The subjective symptoms are a pricking pain, itching, heaviness of the lids, photophobia and some lacrymation.

Treatment.—No remedy has yet been found of service. Protection of the eyes from dust and wind and the use of dark glasses must be enjoined. The cold application on

page 94 will be found comforting. The yellow oxid of mercury ointment may be tried. An ointment of salicylic acid, 2 per cent., rubbed into the conjunctiva once a day has been recommended. This treatment should be preceded by a drop of cocaine solution. One part of dilute acetic acid to 250 of water is said to relieve the pricking pain.

PTERYGIUM.

This is a triangular mass of hypertrophied conjunctiva, the apex of which encroaches upon the cornea, with the base generally toward the inner, sometimes toward the outer

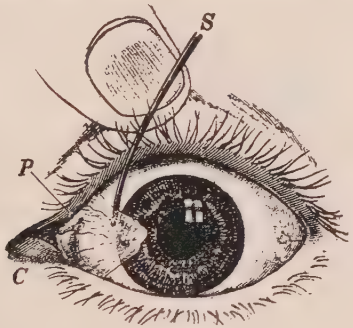

FIG. 83.—Pterygium. C, caruncle; P, punctum; S, probe passed under the upper margin. (Fuchs.)

canthus. In rare instances an eye may have two pterygia, one on each side. The head or apex is firmly united to the cornea, sometimes going deep enough to destroy the membrane of Bowman. A pterygium rarely grows beyond the center of the cornea and usually it requires years for the apex to reach that point. While progressing a pterygium is red, fleshy and vascular (p. crassum), later, development ceases and it becomes thin, white, membranous and more or less bloodless (p. tenue). It affects vision by growing in front of the pupil or by traction producing astigmatism.

Cause.—It is found usually in those whose eyes are subjected to the irritation of wind and weather. Fuchs claims

it is developed from a pinguecula, others maintain that its starting-point is an erosion of the corneal limbus. Laymen will usually call this growth cataract.

Treatment is operative. *False pterygium* partakes of the character of a symblepharon. It is an inflammatory adhesion of the ocular conjunctiva to a denuded or ulcerated point of the corneal limbus which is the result of acute blennorrhea, diphtheria, burns or injury. It can be differentiated from true pterygium by its history, the fact that it may appear at any point on the circumference of the cornea, and that it has no tendency to progress.

Fig. 84.—Symblpeharon.

PINGUECULA.

This is a small yellow elevation in the conjunctiva, generally found between the limbus of the cornea and the plica semilunaris, but sometimes on the temporal side. It is composed of connective tissue and elastic fibres. It is of frequent occurrence, does no harm and need not be removed.

SYMBLEPHARON.

This is a cicatricial adhesion between the conjunctiva of the lid and the conjunctiva of the ball and is the result of the apposition of two raw surfaces, which may have been produced by operations, ulcers, burns, etc. The treatment is operative and difficult. After dissecting the lid from the ball the raw surfaces must be thoroughly covered by mucous membrane or they will promptly reunite.

BURNS.

Burns of the conjunctiva are serious because they lead to the adhesion between the lids and globe just described.

Powder burns may only involve the outside of the lids and may, if the eye is not closed quickly enough, seriously damage the cornea and entail loss of sight. The burns of percussion caps and torpedoes are especially destructive, owing to the added evil of the chemical action of the fulminate of silver and mercury of which they are made. All the foreign particles should be carefully picked out of the skin and cornea, an anodyne given to control the pain, and the eye put up in an aseptic oil dressing. If the cornea is much injured atropin should be used, as there is danger of secondary iritis.

Lime burns must be washed copiously with tepid water and all particles picked out with forceps if an anesthetic has to be given to accomplish it. A weak solution of vinegar may be used. An anodyne can be given and cocain used locally. Adhesions should be broken every day and sweet oil dropped between the lids. If the burn is deep symblepharon will follow.

Acid burns should be thoroughly cleansed with weak bicarbonate of soda solution, and the raw surfaces, pain and inflammation combated as in the case of lime burns. Atropin should always be used where there is danger of iritis.

SUB-CONJUNCTIVAL ECCHYMOSIS.

A hemorrhage under the conjunctiva may be due to a strain, traumatism or disease of the blood vessels. It is seen often in children with whooping cough, and need cause no uneasiness. Coming on in an adult, without strain or accident, it indicates weakness of the vessel walls and portends hemorrhages in other organs, which might be of serious consequence. There is no pain attending the con-

dition and treatment is unnecessary. Hot applications may hasten absorption of the clot.

MORBID GROWTHS IN THE CONJUNCTIVA.

Under this head I will only mention the abnormalities which may develop, as the diagnosis and treatment of these conditions lie along surgical lines. The conjunctiva may be the site of a primary syphilitic sore, or a secondary mucous patch and a tertiary gumma of the conjunctiva has been reported. If a true chancre is present the preauricular and submaxillary glands will be swollen. Treatment is constitutional.

FIG. 85.—Thickening of the lower lid due to a mass of tubercular nodules.

Tuberculosis of the conjunctiva, though a rare disease, is now being more often recognized. It may appear in a number of forms but most frequently occurs as an ulcer. The next most frequent variety is that characterized by yellowish subconjunctival nodules which may be mistaken for trachoma follicles. The detection of conjunctival tuberculosis often requires fine diagnostic discrimination. Epitheliomata and sarcomata may develop, and usually elect the limbus as their starting-point. Thorough removal is imperative. Some cases demand sacrifice of the eyeball and orbital contents, but even this does not always save the

patient. Lipomata are found under the conjunctiva, between the superior and external rectus muscles and must be differentiated from a dislocated lacrymal gland. Papillomata may grow from any part of the conjunctiva, while Dermoid tumors are, as a rule, found as congenital formations, near the outer canthus. Cysts, Nevi and Angiomata are also found in the conjunctiva.

LESSON XVII.

DISEASES OF THE CORNEA.

PHLYCTENULAR KERATITIS.

Phlyctenular keratitis and phlyctenular conjunctivitis are the same disease, the only difference being in the location of the vesicle. The small blister, which is the characteristic feature of the disease, may be located on the scleral conjunctiva or on the cornea, but is most frequently found between the two, at the limbus. When on the cornea, all the symptoms are more severe than when the disease is

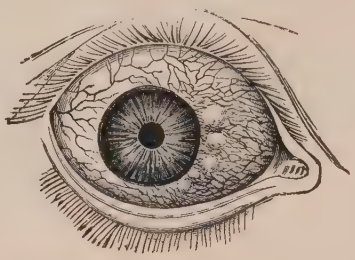

FIG. 86.—Phlyctenular conjunctivitis. (After Dalrymple.)

conjunctival, and it is only when corneal that it can leave any changes which impair vision. The number of vesicles is not limited, and it is possible to have them on the cornea and conjunctiva at the same time.

Symptoms.—The so called vesicle, a small nodule (red if on the conjunctiva, gray if on the cornea), is at first a circumscribed accumulation of leucocytes, under the epithelial layer, but soon develops into an ulcer. The conjunctiva is injected, and there is a tendency of the enlarged vessels toward the phlyctenule. Photophobia and pain are severe, which produces strong blepharospasm. The child will keep

its face in the dark and any effort to bring it to the light, for the purpose of examination, will meet with strong resistance. Lacrymation is pronounced. Marginal blepharitis is often present. Generally there is a discharge from the nose, and eczematous scabs form around the lips and nostrils. There may be enlargement of the lymphatic glands and other evidences of a strumous diathesis. Usually the disease recovers in a few weeks, leaving no permanent injury, but relapses are the rule. Faint opacities of the cornea may be left, which, if over the pupil, will impair vision. In rare instances deep ulceration of the cornea may develop, fol-

FIG. 87.—Phlyctenular keratitis. (After Iwanoff.) This consits of a collection of pus cells between the epithelium and the substantia propria. The band passing obliquely to it is a corneal nerve.

lowed by secondary iritis, perforation or staphyloma.

Cause.—It is a disease of childhood, and is supposed to be due to some irregularity of nutrition, the result of the strumous diathesis. Bad hygienic surroundings and insufficient nourishment seem to contribute to the disease, and yet it is frequently seen in otherwise healthy children.

Treatment.—The cause being constitutional, give fresh air, wholesome food, tonics of syrup of the iodid of iron, malt or cod liver oil, and keep bowels regular. Small doses of calomel are efficacious. Dusting the cornea with finely powdered calomel, once a day, is recommended but must not be done if the patient is taking potassium iodid. Promote health in every way. Use locally hot fomentations

and a weak ointment of yellow oxid of mercury rubbed in gently once a day. Atropin is often indicated. If pronounced corneal ulceration develops, follow the treatment for such a condition (page 122). Constitutional treatment should be continued after the disappearance of the local disease.

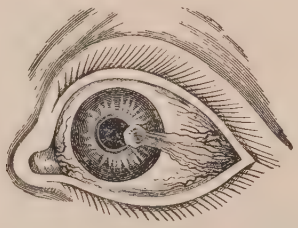

FIG. 88.—Phlyctenular ulcer.

INTERSTITIAL KERATITIS.

This is a disease of childhood but may be found in early adult life. Both eyes are usually involved, one in advance of the other. The substantia propria is the part primarily involved. The deep layers soon participate in the inflammation, and in severe cases the uveal tract rarely escapes. The course

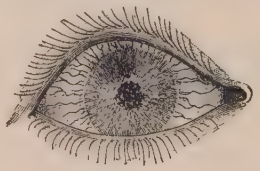

FIG. 89.—Interstitial keratitis. (Nettleship.)

of the disease is chronic, sometimes extending over two or three years. The prognosis is favorable, although only a few cases escape without some impairment of vision. In a limited number sight is permanently lost.

Symptoms.—At first the eye will indicate a state of irritability. There will be some photophobia, lacrymation and circumcorneal hyperemia. Vision will become blurred and

inspection will reveal an infiltration of the deeper layers of the cornea, which gives it an opaque or hazy appearance. This haziness may begin in the center or it may start from the scleral margin. Small blood vessels will be seen springing from the corneal periphery and extending towards its center. These blood vessels are deep in the substantia propria and if numerous will give the inflamed area a salmon pink color. The opacity of the cornea may become complete in a short time and vision be reduced to light percep-

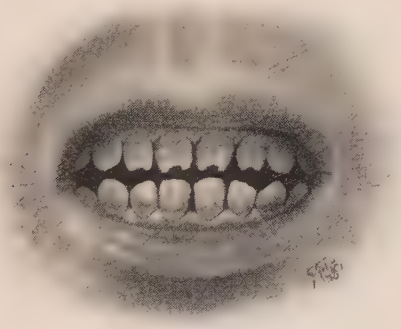

FIG. 90.—Hutchinson's teeth. Also the syphilitic scars at the angles of the mouth.

tion. Iritis may occur, with a tendency toward the inflammation extending to the ciliary body and choroid. When resolution sets in the opacity begins to disappear at the margin, the center of the cornea being the last part to become transparent. When the iris can be seen, posterior synechiae may be found and when the fundus can be examined we may find evidences of choroidal inflammation. Associated with the eye symptoms we generally find evidences of inherited syphilis; glandular enlargement, sunken nose, ozena, Hutchinson's teeth, scars at the angles of the mouth, the vaulted palate and the characteristic physiognomy.

Cause.—Nettleship claims to have found evidences of inherited syphilis in 68 per cent. of his cases, and suspects it in the remaining 32 per cent. It is said also to be caused by scrofula, rheumatism and acquired syphilis.

Treatment.—Use smoked glasses to protect the eyes from the light. Apply hot applications for thirty minutes at a time three or four times a day. Drop into eyes, twice a day, a one per cent. solution of sulfate of atropin. Assume the existence of syphilis and give anti-syphilitic remedies with tonics, good food and good air. After the acute symptoms have subsided use a weak ointment of the yellow oxid of mercury (grs. 2 to ℥ i) putting into the conjunctival sac, once a day, a quantity about half the size of a pea. The ointment can be thoroughly disseminated, and at the same time a massage of the cornea effected, by placing a finger on the closed lid and giving it a gentle lateral or rotary movement. Dusting the cornea with calomel has been recommended, but it must be remembered that calomel should never be put into an eye when the patient is taking iodid of potassium, as the iodid is found in the tears and with the colomel makes an intensely irritating compound, mercuric iodid. If the massage with the yellow oxid of mercury ointment or the dusting with calomel causes undue reaction, it indicates that the remedy has been employed too soon and its use should be postponed until the eyes are less sensitive.

LESSON XVIII.

DISEASES OF THE CORNEA (*Continued*).

ULCERATION OF THE CORNEA.

Symptoms.—Congestion, pain, lacrymation, impairment of vision and swelling of the lids are associated with ulceration of the cornea, but the latter, being due to such a diversity of causes, will show a great variety of symptomatic pictures. For example, an eye with ulceration of the cornea resulting from diphtheritic conjunctivitis will neces-

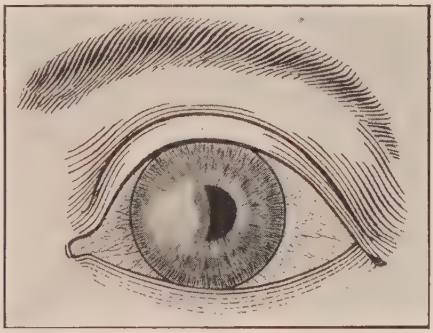

FIG. 91.—A large superficial ulcer of the cornea. The ulcer is surrounded by a zone of infiltration.

sarily present a very different appearance from one in which the ulcer is due to an infected foreign body. Corneal ulcers have also been accurately classified according to shape, method of development and cause, but for the purposes of the student a general description is deemed sufficient.

The part of the cornea involved becomes infiltrated and appears hazy, white or yellow. This is quickly followed by a loss of corneal substance. The destruction of tissue may

spread superficially or may involve the deeper layers and result in speedy perforation. The ulcer will be surrounded by a hazy zone of infiltrated tissue, the region of densest infiltration corresponding to the direction in which the ulcer is most liable to progress. If the disintegrating process only involves the first two layers, repair with transparent tissue will result, but any loss of the deeper layers will be replaced by an opaque scar. An ulcer will meet with greater resistance from the membrane of Descemet than from any other layer of the cornea, and is often checked at this point. If

FIG. 92.—Beginning corneal ulcer. (After Sæmisch.) The upper layers of epithelium are partly lacking. At Bowman's membrane a layer of pus-cells is seen. In the substantia propria are numerous small groups of pus-cells.

Descemet's membrane gives way, perforation follows. When this takes place the aqueous escapes and the iris and lens come forward to the cornea. If the iris adheres permanently to the corneal cicatrix, we have a condition called *anterior synechia.* Contact of the anterior lens capsule with the cornea is liable to produce an opacity of the capsule at the point of contact if the patient be very young. When the aqueous escapes the tension is relieved and the lymph circulation in the cornea becomes freer, which accounts for the improvement so often noted after perforation. Iritis occurs frequently and is sure to occur if the deeper layers of the cornea are involved. Adhesion of the iris to the lens capsule or *posterior synechia* must be guarded against. The ciliary body may become involved. In some cases there is

an exudation of non-pathogenic pus from the iris which forms at the bottom of the anterior chamber. Pus in the anterior chamber is called *hypopyon*. The presence of hypopyon adds gravity to the disease, and in such cases the prognosis should be extremely guarded. The entire cornea may melt away and the eyeball still be preserved by the formation of a white, fibrous cicatrix where the cornea was. This new tissue may not be as resisting as the cornea, and is liable to be protruded by the intra-ocular pressure, causing

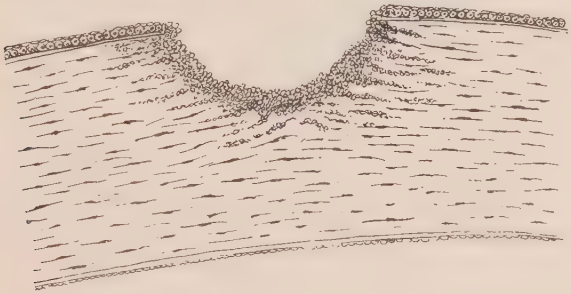

FIG. 93.—Ulcer of the cornea. The epithelium, Bowman's membrane and part of the substantia propria are gone. The floor of the ulcer is infiltrated with pus-cells.

staphyloma (page 126). After perforation intra-ocular infection may occur and the eye be destroyed by *panophthalmitis* (page 149).

Cause.—The *exciting* cause is a pathogenic microbe, generally the white or yellow staphylococci, the pneumococcus or the streptococcus. The source of the germ may be purulent conjunctivitis, dacryocystitis, erysipelas, diphtheria, ozena, septic fingers, handkerchiefs and instruments, or an unknown source.

The *predisposing* cause is some condition which renders the cornea more susceptible to infection. This may be a debilitating disease, an injury from a foreign body, an oper-

ation, lagophthalmia (paralysis of the seventh nerve), or paralysis of the fifth nerve. The ulceration due to paralysis of the fifth is called *neuroparalytic keratitis*. With paralysis of the fifth there is loss of sensation, foreign bodies are no longer removed from the cornea, by the reflex action of the lids, and abrasion results. Abrasion is further faciliated by the dryness of the cornea which exists in the absence of winking.

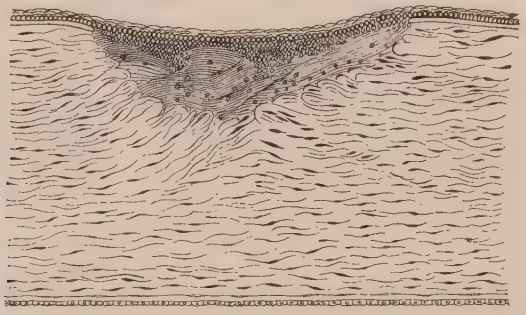

Fig. 94.—The cornea after ulceration, showing the scar tissue.

Treatment.—When the ulcer is due to purulent conjunctivitis, dacryocystitis erysipelas, diphtheria, etc., the primary disease must be treated vigorously. If the secretion is scant the lids should be immobilized, between treatments, by a light bandage, if abundant the bandage should not be used. The focus of germs should be destroyed by touching the ulcer with a galvanic cautery, tincture of iodin or carbolic acid, or by scraping it clean with a small curette. This should be done under holocain (one per cent.) anesthesia. Holocain is preferable to cocain as it does not dry the corneal epithelium, and also possesses some antiseptic properties. The conjunctival sac should be cleansed out about three times a day with bichlorid or cyanid of mercury solution 1 to 8,000. The cleansing may be repeated more fre-

quently if a saturated solution of boracic acid or biborate of soda is used. The direct application of a strong protargol solution (20 per cent.) has been extolled. Argyrol (10 to 40 per cent.) may be used. Covering the ulcer with finely powdered nosophen or xeroform is advised. Hot fomentations should be applied for thirty minutes at a time every

FIG. 95.—Perforating ulcer of the cornea, adhesion of iris (Anterior synechia).

four hours. About three times a day instil a drop of a one per cent. solution of atropin to relieve iritic congestion and prevent posterior synechia. Some advise the use of eserin to relieve intra-ocular tension and thus improve the lymph circulation in the cornea, but

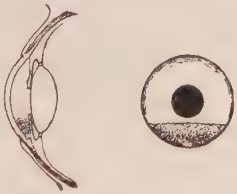

FIG. 96.—Hypopyon, seen from the front, and in section, to show that the pus is behind the cornea. (Nettleship.)

this is manifestly dangerous if the iris is involved. The local application of a 10 per cent. ointment of cassaripe is said to relieve pain and otherwise favorably influence the disease. Paracentesis will relieve the tension and is sometimes indicated, especially if perforation is imminent. The patient should be kept quiet and the constitution sustained by iron, quinin, and strychnin tonics. If the ulceration is neuroparalytic or is due to paralysis of the seventh nerve, the cornea must be protected by the lid and a bandage or adhesive plaster will be necessary to accomplish it.

LESSON XIX.

DISEASES OF THE CORNEA (*Con'd*) AND SCLERA.

VASCULAR KERATITIS OR PANNUS.

The upper half of the cornea is the part most frequently affected, but its whole surface may be involved. It becomes grayish in color from cellular infiltration and covered by a mesh of fine blood vessels, which grow from the conjunctiva. The infiltration and vascularity are found between the epithelial and Bowman's layers, but may go deeper. If the new growth invades the substantia propria permanent scarring is the result. Vision is impaired and may be reduced to

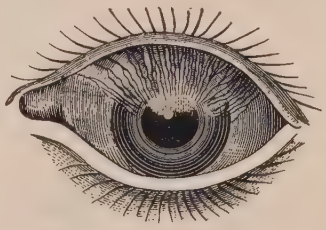

FIG. 97.—Pannus. (Fick.)

light perception. As pannus is secondary to some other ocular disease the general symptoms will be those of the primary affection.

Cause.—Long-continued irritation of the cornea from trachoma, many considering it the only cause of true pannus, persistent phlyctenular keratitis, ingrowing lashes, exposure from imperfect closure of the lids, etc.

Treatment.—Attend to the primary disease. The use of an infusion of jequirity produces a severe and dangerous purulent inflammation, which often results in great improve-

ment and sometimes cure of the pannus, but this treatment should be left to an oculist.

OPACITIES OF THE CORNEA.

Nebula, macula and leucoma are names given to opacities of the cornea. These opacities usually represent scar tissue, which has replaced the loss of substance occasioned by an ulcer, but they may be due to an infiltration or a traumatism.

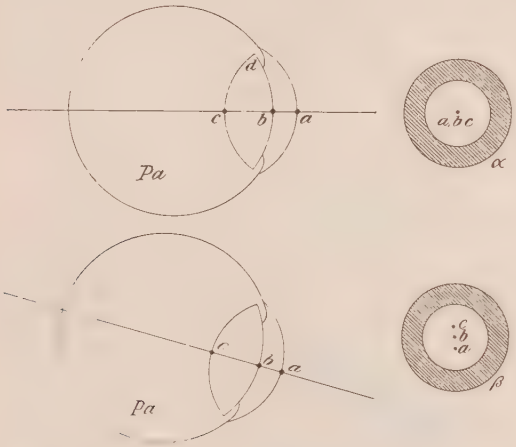

FIG. 98.—Localization of opacities in cornea and lens.

If the first two layers of the cornea are destroyed they may heal without leaving any sign, but any loss of the deeper layers is repaired with scar tissue. The amount of damage to sight produced by an opacity depends upon its location relative to the pupil. Recent scars are improved by time and direct massage with a stimulating ointment, but old ones will remain unchanged. If the opacity is central and there is any peripheral clear cornea, an artificial pupil may improve vision. Before advising an iridectomy it is wise to dilate the pupil to the maximum extent and observe if this measure improves vision; if not, a false pupil will be of little service.

At times vision is much impaired by opacities which are so faint that they may be overlooked by inspection, even with oblique illumination. With an ophthalmoscope and transmitted light they will be seen. The exact locality of such

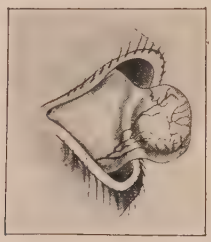

FIG. 99.—Total staphyloma. (Fick.)

an opacity, if in the cornea, anterior or posterior part of the lens, is often not easy to determine (Fig. 98).

STAPHYLOMA.

Severe ulceration of the cornea so decreases its power of resistance that the normal intra-ocular pressure may cause it

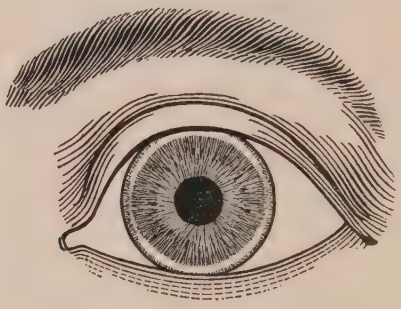

FIG. 100.—An arcus senilis.

to bulge forward, destroying the natural curve. The distension may involve the whole cornea or only part of it. When staphyloma is the result of a perforating ulcer, the iris may be caught in its tissue and severe pain and intra-ocular inflammation result. The staphylomatous cornea is

never transparent. It may be stationary or progressive. It may be small or so large that the lids will not close over it. In some cases nothing need be done. The treatment is operative.

ARCUS SENILIS.

A narrow white ring is often seen near the circumference of the cornea. It is usually found in old people but may occur in the young. It is caused by hyaline degeneration and requires no treatment. It has no influence on the healing of wounds, as for example the incision in cataract operation.

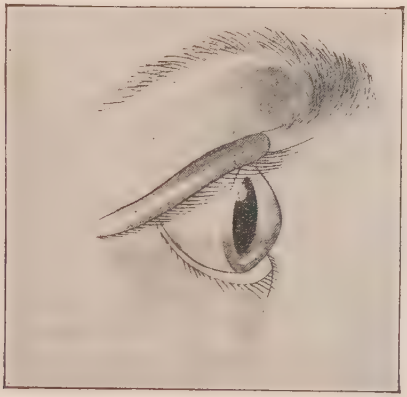

Fig. 101.—Keratoconus. (After A. v. Graefe.)

CONICAL CORNEA.

Sometimes the center of the cornea becomes weakened by an atrophic process and the intra-ocular pressure pushes it forward; the convex sphere changing to a cone. The cornea remains clear except for the occasional appearance of a nebula at its apex. The process is slow and gradual but finally reaches a point where it stops. Vision is greatly impaired. Inspection of the eye reveals no abnormality,

except in pronounced cases, when a side view will show its conical form. Diagnosis in the early stages is difficult and treatment not very effective. The latter should be left to an oculist.

Fig. 102.—Spud for removing foreign bodies.

FOREIGN BODIES IN THE CORNEA.

It is very common for cinders, sand, pieces of emory, iron, steel, etc., to become lodged in the cornea. Pain and lacrymation will be intense, with more or less circumcorneal injection. If simple inspection does not reveal the offender

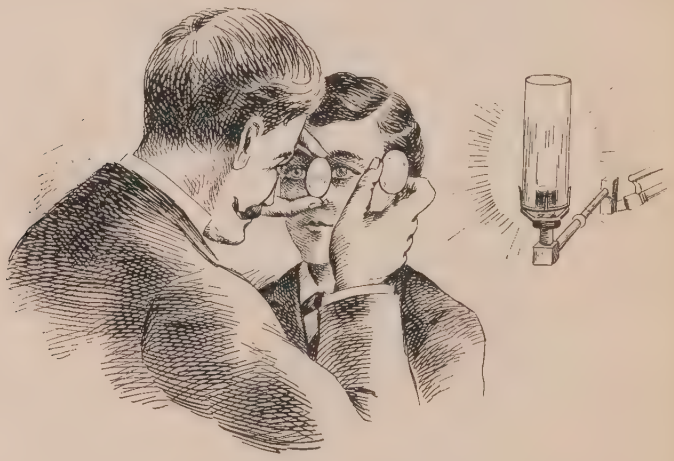

Fig. 103.—Oblique illumination.

use oblique illumination. This is done by seating the patient about two feet from a light and with a 16 or 20 Diopter convex lens, focus the rays *obliquely* on the part to be examined. Then by viewing the illuminated area through a magnifier the cornea, iris and anterior part of the lens may be thoroughly inspected. To remove a foreign body, the

cornea should first be anesthetized by several drops of a 4 per cent. solution of cocain or a 1 per cent. solution of holocain; then, with a needle or spud pick it out with as little destruction to corneal tissue as possible. When the epithelium is denuded there is always danger of infection; therefore, an antiseptic collyrium (solution hydrarg. bichlorid 1 to 8,000) should be used for three or four days, or until the epithelial layer is restored. Another way to prevent infection of the cornea when the epithelium has been denuded, is to touch the lesion lightly with compound tincture of benzoin. A thin, adherent pellicle is immediately formed, which covers the wound for from six to twelve hours.

DISEASES OF THE SCLERA.

EPISCLERITIS.

Under the ocular conjunctiva is a delicate membrane, the capsule of Tenon, and between the capsule of Tenon and the sclera proper is the loose connective tissue called the episclera. These parts are so intimately related that inflammation of the subconjunctival tissues generally involves the overlying conjunctiva and may go deeper into the sclera proper. Scleral and episcleral inflammation is limited to the region anterior to the equator.

Symptoms.—In episcleritis there appears a patch of dusky red injection under the conjunctiva, generally between the insertion of a rectus muscle and the cornea. There may be a distinct nodule which will tend to confound the disease with phlyctenular conjunctivitis. The age of the patient and the fact that the episcleral nodule does not ulcerate will aid in the differentiation. The discharge from the eye is watery and pain and photophobia are generally slight. The inflamed spot may disappear spontaneously, may persist for weeks, has a tendency to recur, and will often leave a gray, discolored patch.

Cause.—Rheumatism, gout, scrofula, syphilis and menstrual derangement. It may arise from exposure to the weather and is also said to appear over the insertion of a rectus muscle suffering from insufficiency. Frequently the cause is obscure.

Treatment.—Some cases are so mild as to need no treatment. The constitutional cause, if discoverable, should be attended to. Correct muscular anomalies and refractive errors. Apply hot fomentation. Use atropin if there is any tendency toward iritis. When chronic, stimulation with yellow oxid of mercury ointment is useful.

Fig. 104.—Staphyloma of the sclera.

SCLERITIS.

Inflammation of the sclera may be circumscribed or diffuse. It resembles episcleritis but the symptoms are all more severe and, as a deeper structure is inflamed, there is much greater danger of involvement of the uvea. The discharge is watery and pain and photophobia may be pronounced. The inflammation may extend to the underlying uveal tract and produce iritis, cyclitis or choroiditis; or extend to the cornea, producing a haziness of its deep layers (sclero keratitis). These complications may lead to an impairment or total loss of vision. Tension is often increased. The condition is chronic in its course, sometimes

extending over a period of years. The scleral wall may become so thinned that the dark uvea showing through it will give it a purplish hue. The imperfect resistance of the thinned sclera will result in bulging or staphyloma of its weakest parts.

Cause.—The cause is generally rheumatism, gout, syphilis, scrofula or menstrual disorders.

Treatment.—For the syphilitic form use mercury and iodid of potassium. In scrofulous cases, tonics, good air and good food. When due to rheumatism, salicylate of sodium, Rochelle salts, etc. If gouty in origin, iodid of potassium or colchicum. Combine above treatment with hot baths, warm fomentations over eyes, leeching of the temples, and atropin locally. The increased tension and staphyloma may be improved by a wide iridectomy.

LESSON XX.

DISEASES OF THE IRIS.

Mydriasis or dilatation of the pupil may be due to many causes, among which are:—

1. The use of drugs called mydriatics, such as atropin, homatropin, scopolamin and cocain. Most mydriatics also produce paralysis of accommodation.
2. Increase of intra-ocular pressure as in glaucoma.
3. Loss of vision as in atrophy of the optic nerve.
4. Paralysis of the third nerve.
5. Dimness of light.
6. Ingestion of certain drugs, belladonna, ergot, etc.
7. Apoplexy in the later stages.

Myosis or contraction of the pupil may be due to:—

1. The use of drugs called myotics, such as eserin and pilocarpin. The myotics also stimulate accommodation.
2. Evacuation of the aqueous humor.
3. Hyperemia of the iris as in iritis.
4. Paralysis of the cervical sympathetic nerve.
5. Bright light, accommodation and convergence.
6. Ingestion of certain drugs, as opium and alcohol.
7. Apoplexy in the early stages.

The Argyll-Robertson pupil is one which responds to convergence but not to light, and is significant of locomotor ataxia. The variations of mydriasis and myosis dependent upon irritation and disease of the brain and spinal cord are too complex to dwell upon here.

Anterior synechia is an adhesion of the iris to the cornea, due to perforation of the cornea and lodgment of the iris in the wound.

Posterior synechia is an adhesion of the iris to the anterior capsule of the lens. In complete posterior synechia we have what is called *exclusion* of the pupil. Where the pupilliary area is filled by a membrane, we have *occlusion* of the pupil (Fig. 107).

IRITIS.

The disease may be divided by its course into acute or chronic; pathologically it may be plastic, suppurative or serous; etiologically it may be divided into as many forms as there are causes, the leading varieties being syphilitic,

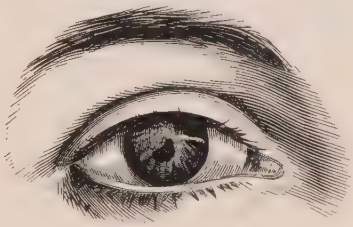

FIG. 105.—Posterior synechia.

rheumatic, gouty, idiopathic, traumatic and secondary. The typical form of iritis, is plastic; serous iritis, according to Collins, Priestley Smith and others, being more appropriately a cyclitis.

PLASTIC IRITIS.

Symptoms.—Injection of the deep blood vessels around the cornea, later extending over the entire white of the eye. Discharge of a watery character. Intolerance of light and pain of a neuralgic nature, beginning in the eye-ball and extending over the brow, temple and cheek. The pupil becomes small and will not react to light. Its normal color changes to a darker tone, a blue or gray iris becoming green. The aqueous becomes turbid from lymphoid cells, pus and red blood corpuscles, and vision is correspondingly impaired. Adhesion will take place between the iris and anterior lens

capsule, constituting posterior synechia. If these adhesions are broken, pigmant deposits will be left on the capsule of the lens. When the attack is syphilitic in origin, gummata may develop on the iris. If there is much pus in the aqueous humor it may settle in the anterior chamber, producing hypopion.

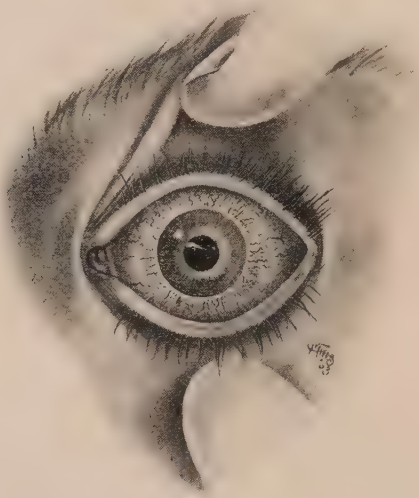

FIG. 106.—Congestion of iritis. The circumcorneal zone the first part to become injection. See Fig. 74.

Sometimes the exudation in the anterior chamber leaves a membrane across the pupil which may be mistaken for cataract. Such a condition is spoken of as occlusion of the pupil (Fig. 107). Iritis may attack one eye or both. Its duration depends largely upon the treatment, but will generally last from two to six weeks. In some cases the eye will be myopic for weeks after an attack of iritis.

Cause.—In fifty per cent. of all cases it is due to syphilis, secondary, tertiary, acquired or inherited. The next most potent factors are rheumatism and gout. It may arise as

secondary to other eye diseases or be due to direct lesion, accidental or operative. Gonorrhoea and diabetes are also said to be etiological factors.

Treatment.—Prohibit work and protect eyes with shaded glasses. Look to the general health of the patient, paying special attention to the condition of the alimentary canal. To prevent posterior synechia, dilate the pupil with atropin and keep it dilated through the whole attack. Leeching at the temple is sometimes efficacious. For the pain, give antipyrin or morphin and apply heat, dry or in the form of

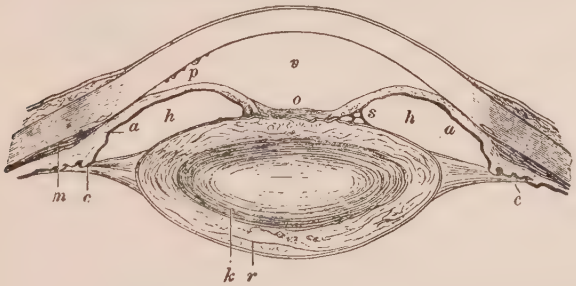

FIG. 107.—Exclusion and occlusion of the pupil, with bulging of the iris forward from accumulation of fluid in the posterior chamber. The posterior chamber (h) is thus made deeper, the anterior chamber (v) shallower, especially where the root of the iris (a) is pressed against the cornea. The pupil is closed by an exudate membrane o. (Fuchs.)

watery fomentation. The daily instillation of several drops of a 2 per cent. solution of dionin has a decided influence in lessening the pain and also seems to shorten the duration of the attack. When dropped on the eye it causes a burning sensation which can be prevented by preceding it with a drop or two of cocain. In syphilitic cases give mercury and iodid of potassium. When rheumatic or gouty in origin, use the salicylates, colchicum, lithia, etc., combined with hot baths or pilocarpin sweats.

SUPPURATIVE IRITIS.

This form is generally due to wounds or operations and does not differ materially from the plastic form, except that

the presence of pus infection makes the symptoms more severe and the prognosis very grave. It may also be due to infectious diseases, pyemia and meningitis.

SEROUS IRITIS.

This disease and serous cyclitis are the same; not only are the iris, ciliary body and the choroid involved, but also Descemet's membrane of the cornea. It has been described under the names descemitis, keratitis postica, keratitis punctata, serous uveitis, and serous irido-cyclitis.

Symptoms.—Slight pericorneal injection, pain insignificant, vision sometimes only a little below normal, but may be much lowered, increase of aqueous evidenced by unusual

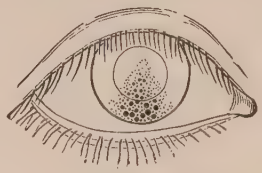

FIG. 108.—Serous iritis.

depth of the anterior chamber and plus tension. The pupil will not be contracted as in plastic iritis and the iris will only be slightly discolored. Posterior synechia may occur, but is not as common as in other forms of iritis. There will also be found a characteristic cellular deposit in the form of fine dots on the lower half of the posterior surface of the cornea, which constitutes *keratitis punctata.* The course of the disease is more or less chronic and the subjective symptoms mild as compared with the other forms of iritis.

Cause.—The causes are the same as in plastic iritis.

Treatment.—The same as in plastic iritis except that atropin must be carefully used owing to the danger of increasing the tension. If the tension becomes dangerous it may be reduced by the cautious use of pilocarpin locally, or pilocarpin injections, to produce diaphoresis.

LESSON XXI.

DISEASES OF THE CILIARY BODY AND VITREOUS.

DISEASES OF THE CILIARY BODY.

Inflammation of the ciliary body is not an isolated condition, but is probably always associated with disease of the iris or choroid. Cyclitis may be acute or chronic; plastic, suppurative or serous.

PLASTIC AND SUPPURATIVE CYCLITIS.

The symptoms of these two conditions are the same as in iritis, with the addition of opacity of the vitreous, severe pain upon pressure over the region of the ciliary body and characteristic tension, which is plus in the acute stage but later may become decidedly minus, due to atrophy of the ciliary body and shrinkage of the vitreous. The lens sometimes becomes opaque and detachment of the retina may occur. Plastic cyclitis is dangerous, suppurative cyclitis is almost always fatal to vision. Treatment is the same as in iritis.

SEROUS CYCLITIS.

This is the same as serous iritis (page 136).

SYMPATHETIC OPHTHALMIA.

Sympathetic ophthalmia is a diseased condition arising in one eye, caused by some organic lesion of its fellow. The eye which is first affected is called the *exciting* eye, while the other is called the *sympathizing* eye.

The disease takes two forms—sympathetic irritation and sympathetic inflammation.

Symptoms.—(1) Sympathetic irritation is a functional derangement characterized by intolerance of light, lacrymation and fatigue of the eye when used. Visual acuity may be impaired and sometimes temporary obscuration of sight occurs. There may be considerable pain, of a neuralgic character, in and around the eye, and also some pericorneal injection. The symptoms may subside, but a relapse will occur. Unless the exciting eye is enucleated, the disease is prone to develop into sympathetic inflammation.

(2) Sympathetic inflammation is sometimes very slow and insidious in its development. When established there is intense circumcorneal injection, an inflamed iris, contracted pupil, punctate deposits upon Descemet's membrane, lowered vision, opacities in the vitreous, intense neuralgic pain in the region supplied by the fifth nerve; also pain upon pressure over the ciliary region. The iris, ciliary body and choroid are involved in a chronic plastic uveitis which usually results in total blindness. As the disease progresses optic neuritis, synchisis of the vitreous, detachment of the retina and atrophy of the ball will develop.

Cause.—The cause is an inflammation of the uveal tract of the exciting eye. The uveitis may be idiopathic, but the inflammation most prone to excite sympathetic trouble is that due to a wound of the ciliary region or the presence of a foreign body in the exciting eye. Other sources of the exciting uveitis are perforating corneal ulcers and intraocular tumors. After an enucleation the optic nerve or ciliary nerves being caught in the cicatrix have been known to give rise to sympathetic irritation. Sympathetic ophthalmia may arise at any time from two weeks to many years after the lesion of the exciting eye. In spite of many theories our knowledge of how this inflammation is conveyed from one eye to the other is yet speculative.

Treatment.—As sympathetic irritation is always cured by enucleating the exciting eye, this should be done at once, but if sympathetic inflammation is established this procedure will rarely stop it and should not be resorted to if the exciting eye has useful vision, as it will often retain the best vision of the two. If, in sympathetic inflammation, the exciting eye is blind, enucleate it. Its removal may do some good and can do no harm. The patient should be kept in a dark room, hot fomentations used from four to eight hours a day, anodynes given for pain and mercury and tonics given internally. Atropin and dionin should be used as in plastic iritis. Salicylate of soda to the limit of toleration has been very successful in some cases. As the treatment of sympathetic inflammation is so unsatisfactory, its prophylaxis becomes doubly important and I would advise the enucleation of all blind eyes affected with chronic irido-cyclitis; all eyes with irido-cyclitis due to the presence of a foreign body, which cannot be removed, even if some vision remains; also all shrunken globes and stumps which are tender on pressure.

DISEASES OF THE VITREOUS.

MUSCAE VOLITANTES.

The vitreous consists of 98.5 per cent. water and 1.5 per cent. of a reticulated frame work of very fine fibers, within the meshes of which are found connective tissue cells and migratory leucocytes. These normal cells of the vitreous sometimes become so apparent as to cause considerable annoyance. They appear as spots in front of the eye which may assume a great variety of shapes. They are most apparent when the patient looks toward some bright background such as the blue sky or a sheet of white paper. Vision will be normal and an examination with the ophthalmoscope will reveal no opacities or any other pathologic

condition. It is difficult to explain why the shadow of these fixed cells will give more annoyance at one time than another. These periods of annoyance seem to depend, in a measure, upon faulty digestion and patients will often associate them with "biliousness." The treatment consists in correcting any refractive error and any defect in digestion, also impressing the patient with the harmlessness of the condition and urging upon him the necessity for disregarding the symptoms.

OPACITIES OF THE VITREOUS.

Vitreous opacities may appear as fine dust, floccuh, threads, flakes, large masses or membranes. If the vitreous

FIG. 109.—Opacities of the vitreous, dust like at the lower part of the pupil, with threads and membranous masses above. (Jackson.)

has become fluid (synchisis) the opacities will change their position with every movement of the eye.

Symptoms.—The patient will complain of lowered vision which will be found relatively worse for distance than for near. He will see spots in the field of vision which correspond in size, shape and position with the opacities which cause them. If the vitreous is fluid, vision will be best when the eye is kept still long enough to allow the opacities to settle. Opacities of the vitreous can only be seen objectively with the ophthalmoscope.

Cause.—The opacities are inflammatory exudates or hemorrhages which originate in diseases of the ciliary body,

choroid or retina. The fine dust-like opacities found in the posterior part of the vitreous are due to syphilitic involvement of the retina and choroid. Large black masses are often caused by the choroiditis, which is associated with high degrees of myopia. Gout, tuberculosis, malaria and senility are considered predisposing causes.

Treatment.—Small and recent opacities may be cleared up, but if they are large or of long standing there is little prospect of a cure. Diaphoresis induced by pilocarpin injections is advised. Also purging with salines. Dionin is a powerful local lymphagogue and might prove of value. Main reliance is placed in the constitutional treatment and mercury and potassium iodid have proven the best remedies we possess.

HEMORRHAGE INTO THE VITREOUS.

Symptoms.—If the hemorrhage is small the result will be a clot in the vitreous amounting to an opacity, the symptoms of which have already been given. Vision will depend upon the amount of blood emptied into the vitreous. If the hemorrhage is pronounced vision will be reduced to the perception of light and the light will appear red. At times even light perception is lost. When the vitreous is permeated with blood the diagnostic value of the ophthalmoscope is lost, as everything beyond the lens appears black. The iris and ciliary body may become inflamed and glaucoma is not an infrequent result.

Cause.—Rupture of a blood vessel of the ciliary body, choroid or retina from traumatism or disease.

Treatment.—If the hemorrhage seems to be spontaneous, absolute quiet and the internal administration of such remedies as gallic acid or ergot may be of service. An effort should be made to produce absorption of the clot, to which purpose the treatment for vitreous opacities is applicable. If the hemorrhage is profuse the prospect is not flattering.

FOREIGN BODIES IN THE VITREOUS.

Foreign bodies such as splinters of wood, lead shot, small stones, particles of metal and fragments of glass, may be accidentally driven into the vitreous. The wound of entrance may be destructively large or so insignificant that it is hard to find. It is often very difficult to determine positively that a foreign body is present. The X-ray is of great value in such cases. As a general proposition it may be said that the foreign body or the eye must be removed. Particles of iron and steel may be removed by the electro-magnet. A diamagnetic body must be withdrawn with forceps or carried out with the vitreous which escapes through an incision made as near as possible to the point where the foreign body is lodged.

LESSON XXII.

DISEASES OF THE CHOROID.

The function of the choroid is to nourish the retina and vitreous, and to prevent reflection, by the power to absorb light possessed by its pigment. In albinos there is almost a total absence of pigment in the uveal tract, and great distress from photophobia is the result. Albinos are, as a rule, afflicted with amblyopia, refractive errors or nystagmus. Dark glasses are often a necessity to these patients.

CHOROIDITIS.

Choroiditis is either plastic or purulent. The plastic form is divided into disseminate, central, syphilitic choroido-retinitis and myopic choroiditis. When purulent choroiditis results in inflammation of all the structures of the eye, and this is its usual termination, it is called *panophthalmitis.*

PLASTIC OR EXUDATIVE CHOROIDITIS.

Symptoms.—If the morbid process is limited to the choroid, external signs of inflammation are absent. Visual disturbance will be the only subjective symptom. The objective symptoms are revealed by the ophthalmoscope. There may be one or more areas in the field of vision in which objects are seen dimly or not at all. These areas are called scotomata. If vision is only diminished in a scotoma it is said to be *relative,* if it is entirely absent the scotoma is *positive.* Vision may be reduced by a diffuse cloudiness of the vitreous, or if opacities have formed in it there may be movable dark spots in the field. Night blindness is a frequent symptom (page 185). Patients may complain of sparks or lights before their eyes. There may also be dis-

tortion of the outline of objects called *metamorphopsia.* For example, in metamorphopsia a series of parallel lines will appear wavy in places, or when reading, a word in a line will appear above or below its normal position. If the choroidal exudate causes a separation of the percipient elements of the retina objects will appear unnaturally small, a condition called *micropsia. Megalopsia,* a condition in which objects look unnaturally large, is produced by shrinkage of the exudate. If the inflammatory process is limited to the periphery of the choroid there may be no subjective symptoms whatever. The visual disturbance will depend upon the proximity of the inflamed area to the macula and the amount of involvement of the vitreous.

The ophthalmoscope will show, in recent cases, ill-defined yellowish patches under the retinal vessels. These spots of exudation may absorb and leave no sign, but generally the choroid at these points atrophies and the sclera shows glistening white through it. Around the borders of these atrophic areas, pigment is soon deposited. The retina over the inflamed area is usually involved and also partakes of the subsequent atrophy, which explains the scotomata or blind spots in the field of vision. The inflammatory process may extend to the vitreous and cause a cloudiness of that body, at times so dense as to prevent an ophthalmoscopic view of the underlying tissues.

Cause.—Syphilis, malnutrition, scrofula, anemia, a blow upon the eyeball, high myopia, and in some cases no cause can be assigned with any degree of accuracy. The choroid, being a part of the uveal tract, is subject to inflammation arising in the iris and ciliary body.

Treatment.—Absolute rest of eyes and the use of mydriatics, smoked glasses, mercury, iodid of potassium and tonics.

Disseminate choroiditis is the form inflammation of the choroid is most likely to assume. As the name indicates,

there is a general involvement of the choroid. There will be a number of areas of exudation or atrophy, depending upon the stage of the disease, scattered over the fundus. As long as the macular region is not involved central vision may remain good. Therefore disseminate choroiditis may be far advanced before the patient is conscious of any eye trouble. Its course is chronic. Treatment may check the disease but cannot restore the function of the atrophic areas.

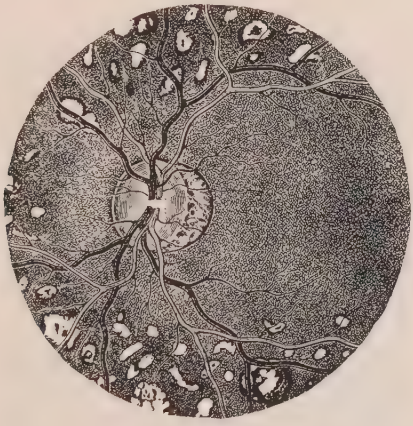

FIG. 110.—Choroiditis disseminata. The macular region not being involved central vision might be normal. (After De Wecker.)

Central choroiditis involves the macular region. It may consist of one large inflammatory area or several smaller ones. Direct vision is impaired or lost and there is a central scotoma in the field of vision. Central choroiditis is often due to myopia of high degree or to senility. The discovery of a central choroiditis after the removal of a cataract is a disagreeable surprise to patient and operator.

Syphilitic choroido-retinitis is a diffuse inflammation of the choroid and retina. The fundus involvement is both central and peripheral. Whether the disease is primarily choroidal or retinal is an open question. The retina will

appear opaque and edematous and under it can be seen the numerous yellow spots characteristic of choroidal exudation. A fine dust-like opacity in the posterior part of the vitreous is usually present. It may be so dense as to prevent a clear view of the fundus and also interfere with the patient's vision. If seen early the disease will respond satisfactorily to vigorous mercurial medication. In the later stages, when there are atrophic spots in the choroid and retina, treatment can at best only check the disease.

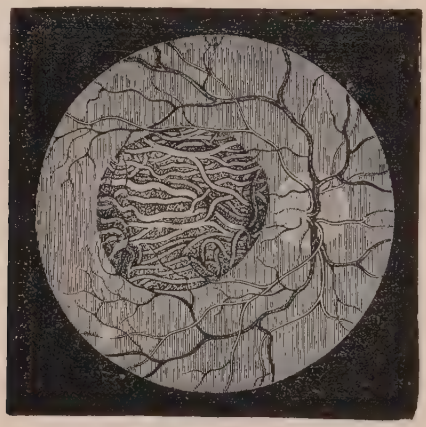

FIG. 111.—Central choroiditis. (Wecker and Jaeger.)

Myopic choroiditis presents two characteristic pictures. One or both may be present. They are: (1) A well defined white crescent on the temporal side of the disc, and (2), central choroiditis. The elongation of the antero-posterior axis found in myopia, particularly in the progressive form, is due to a protrusion backward of the sclera called *posterior staphyloma.* This bulging of the sclera stretches the choroid at the temporal side of the disc. The atrophy of the choroid which follows allows the white sclera to show through, producing the "myopic crescent." At

times the atrophic area encircles the disc but it is most prominent on the temporal side. When the margin of the crescent is sharply outlined and pigmented it usually indicates that the myopia has ceased progressing.

Myopia of high degree will usually produce choroidal changes other than the myopic crescent. These exudative and atrophic spots may be found in any part of the fundus but they are almost invariably located at the macular region. See central choroiditis, page 145.

FIG. 112.—Atrophy after syphilitic choroiditis, showing various degrees of wasting. a, Atrophy of pigment epithelium; b, Atrophy of epithelium and chorio-capillaris; the large vessels exposed; c, Spots of complete atrophy, many with pigment accumulation.

PURULENT CHOROIDITIS.

Cases of purulent choroiditis may present widely different symptomatic pictures, varying from an asthenic form which develops without the consciousness of the patient (pseudo-glioma) to a violent and excruciatingly painful variety (panophthalmitis). The symptoms common to all cases are suppuration of the choroid, total loss of vision and shrinkage of the eye ball.

In rare instances, particularly in children, suppuration of the choroid may not be attended by any external evidences of inflammation. The first knowledge of the disease may be furnished by the appearance of a yellow reflex from the pupil, which is usually dilated. This reflex is from pus in the vitreous. The condition is called pseudo-glioma and is differentiated from true glioma in the early stage, with difficulty. Vision is permanently lost and shrinkage of the ball follows.

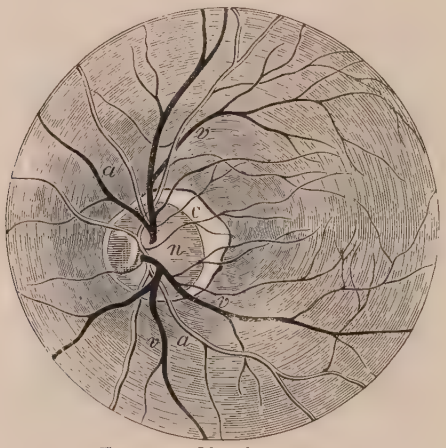

Fig. 113.—Myopic crescent.

The symptoms usually manifested by purulent choroiditis are swelling of the lids, chemosis of the conjunctiva, early loss of vision, severe pain and probably a rise in temperature. If the process begins in an ulcer or wound of the cornea this tissue will soon become opaque and suppurative. If the lesion starts from within, the iris will change color as in iritis, and the aqueous become muddy, but in spite of this the yellow reflex caused by pus behind the lens may generally be seen. In a few weeks the severe inflammation subsides, tension of the ball becomes minus and atrophy begins.

In the severest form (panophthalmitis), all of the above symptoms increase in intensity. The pain is excruciating. There is rise of temperature with vomiting. Tenon's capsule becomes inflamed and thickened, causing exophthalmos and loss of motion. The eyeball will perforate and the pus escape. When this happens the pain and other symptoms rapidly subside to be followed by shrinkage of the ball to a small stump (phthisis bulbi).

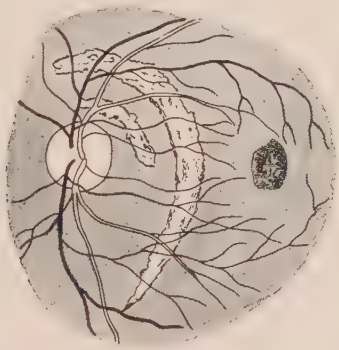

FIG. 114.—Two ruptures in the choroid. (After Knapp.) The retinal vessels pass unbroken across the rupture. The dark spot to the right is a hemorrhage.

Cause.—Intra-ocular pus infection from wounds, operations, perforating ulcers of the cornea, pyemia by metastasis or meningitis.

Treatment.—Control the pain by leeches, hot bichlorid fomentations and anodynes, and enucleate as soon as convinced that the eye is lost. Evisceration is preferred by some as offering less danger of setting up a meningitis than enucleation. If thorough evisceration is not deemed advisable a simple incision through the sclera, to let out the pus, may be made and enucleation postponed until the inflammatory symptoms have subsided.

RUPTURE OF THE CHOROID.

A blow upon the eye may cause a rupture of the choroid. Hemorrhage into the vitreous will usually produce a temporary loss of vision and prevent a view of the fundus. When the blood absorbs a long white crescent-shaped streak will be found usually not far from the temporal side of the disc. If the retina is not injured its blood vessels will course normally over the lesion in the choroid. Permanent loss of vision will depend upon the proximity of the rupture to the macular region.

LESSON XXIII.

GLAUCOMA.

The vitreous and crystalline lens being non-vascular bodies, are nourished by lymph. This lymph is secreted by the choroid, the vascular part of the ciliary body and the posterior surface of the iris. The process of secretion being continuous, there must be a coincident excretion, and we find this takes place in the angle of the anterior chamber, through the spaces of Fontana, which connect with the canal of Schlemm. The direction of the current is from the vitreous, around the lens, into the posterior aqueous chamber, thence through the pupil into the anterior aqueous chamber, thence through the spaces of Fontana and canal of Schlemm into the anterior ciliary veins. Normal intra-ocular tension is dependent upon the maintenance of a physiologic balance between the secretion and excretion of the aqueous humor.

The normal resistance of the globe, when palpated by the index finger of each hand, is designated by the letters Tn. If the globe is abnormally hard its increased tension is designated relatively by the signs T + ?, T + 1, T + 2, and T + 3. If abnormally soft, by T — ?, T — 1, T — 2, and T — 3.

Glaucoma is a diseased condition, due to excessive intra-ocular pressure. It may be idiopathic or secondary to some other pathologic condition of the eye. Idiopathic or primary glaucoma may be simple or inflammatory. The inflammatory form is usually divided into acute, sub-acute, and chronic varieties. All forms of primary glaucoma are pathologically the same disease, the different aspects presented being due to different degrees of intensity. I will therefore

give the symptoms of a mild form (simple glaucoma), and a severe manifestation (acute inflammatory glaucoma); it being understood that the disease may assume innumerable types, varying in intensity, between the two. It must be remembered that glaucoma is generally a disease of relapses and remissions which will always eventuate in total blindness. Both eyes are usually involved, but one may be attacked months or even years before the other.

FIG. 115.—Ophthalmoscopic appearance of a glaucomatous excavation of the disc.

Symptoms of simple glaucoma.—The patient will probably first notice a failure in vision for near work—an unnatural presbyopia, which will necessitate the use of convex lenses at an unusually early age, or require lenses stronger than the age of the patient would naturally demand. Artificial lights may be surrounded by halos or colored rings. At times a mist seems to obscure vision, and occasionally the patient will find himself in total darkness for several seconds. A slight dull pain of a neuralgic character may or may not be felt in and around the ball. The eye generally appears normal except for a slight dilatation of the pupil, the possible

existence of an unusual whiteness of the sclera and the presence of a few large and tortuous conjunctival vessels. In the early stages visual acuity may or may not be reduced, but the field of vision will most likely show peripheral contraction, greatest on the nasal side, and scotoma may exist. Tension of the ball will be increased, with periodic variations in degree, at times approximating so closely to the normal that its glaucomatous nature may not be appreciable.

If the disease has existed for some time, the characteristic cupping or excavation of the optic disc will be seen. If ten-

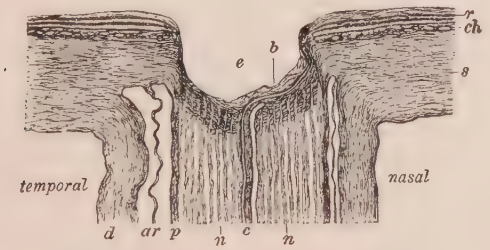

FIG. 116.—Longitudinal section through a glaucomatous optic nerve. Deep excavation, e; remains of nerve fibers, b; choroid, ch; sclera, s; nerve fibers, n; pia mater, p; arachnoid, ar; dura mater, d; central artery of the retina, c; retina, r. (Fuchs.)

sion is appreciably high, there may be spontaneous pulsation of the retinal arteries. Patients suffering with simple glaucoma often consult a physican *only* because vision is impaired and are unconscious of the presence of any pathologic condition. Simple glaucoma may slowly deprive its victim of sight without manifesting any symptoms more active than those detailed, but it frequently changes into the inflammatory type. Simple glaucoma always attacks both eyes, but rarely at the same time. It sometimes occurs in young people and may be found in myopic eyes, which seem to be more or less immune to inflammatory glaucoma.

Symptoms of acute inflammatory glaucoma.—The attack comes on suddenly and, as a rule, at night. It may or may not have been preceded by premonitory symptoms, such as failure of vision for near work, the presence of colored rings around lights, and temporary obscurations of sight. The true nature of the attack may be overlooked, owing to the severe pains in the head and face and the probable presence of an increase in pulse and temperature with vomiting. It may be mistaken for a severe "bilious attack," the inflamed eye being considered incidental. Tension of the globe is

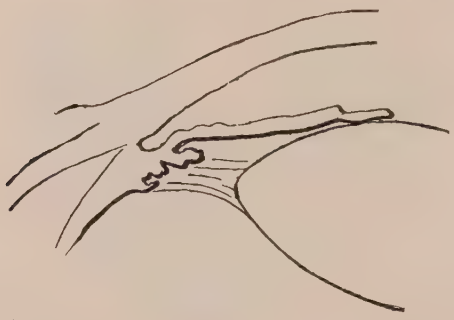

FIG. 117.—Angle of the anterior chamber in the normal eye. (Birnbacher.)

markedly increased, the lids edematous, the conjunctiva injected and chemotic. The cornea will be more or less opaque or steamy and anesthetic, the anterior chamber shallow and its contents possibly cloudy from the presence of lymphoid cells. The pupil will be widely dilated, and the normal black reflex may be green. The interior of the eye will probably not be visible, owing to the opacity of the cornea, but if the fundus can be viewed the veins will appear distended and sinuous and the arteries will be small and show pulsation. If glaucoma has not previously existed the disc will probably not be excavated but it will appear soon if the high tension continues. Vision will of course be greatly reduced. The symptoms may be so intense as to

destroy the eye in a few hours in which case the disease is called *glaucoma fulminans*. As a rule, the symptoms abate and the eye may return almost to the normal in appearance, without however a return of the pre-existing visual acuity. After the lapse of a variable period another attack supervenes, resulting in an additional loss of vision, and so the disease progresses until the absolute state is established and blindness results.

In *absolute glaucoma* the ball is hard, pain constant and intense, the lens cataractous and pushed forward. The

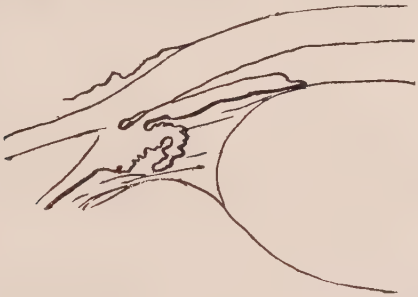

FIG. 118.—Angle of the anterior chamber in glaucoma closed by adhesion of iris base to the periphery of the cornea. (Birnbacher.)

pupil is widely dilated and fixed. The sclera is bluish in color, with a dusky, red, circumcorneal zone, caused by engorgement of the anterior ciliary veins. The cornea is lustreless and vision—even light perception—is gone.

Further changes through which the eye may pass are *degenerative*. There may be ulceration of the cornea with perforation, followed by panophthalmitis and phthisis bulbi. The sclera may give way and staphyloma result; or the eye may slowly atrophy as a result of choroiditis, changes in the vitreous and detachment of the retina.

Cause.—In general terms hypersecretion or sub-normal excretion of the intra-ocular fluid are responsible for the train of symptoms called glaucoma. Blocking up the angle

of the anterior chamber (Fig. 118) by diminishing excretion, is undoubtedly a potent factor in the production of the disease. The use of a mydriatic, by dilating the pupil, pushes the iris into the filtration angle, and will sometimes bring on an attack. The iris is supposed to be forced forward in such a way as to partially block the spaces of Fontana, when a large lens is associated with a small hyperopic eye. That this has something to do with the production of glaucoma seems plausible when we consider that the lens continues to grow until the 65th year and that about seventy per cent. of all cases occur after the age of fifty and the same per cent. are found in hyperopic eyes. An exacerbation may be excited by fatigue, grief, worry or anything which lowers vitality. Among the supposed etiological factors heredity, rheumatism and disturbances of circulation may be mentioned. Secondary glaucoma may be directly attributable to intra-ocular hemorrhage, complete anterior or posterior synechia, traumatic cataract with rapid swelling of the lens, intra-ocular tumors, etc.

Treatment.—As soon as positive of the diagnosis, do a broad iridectomy. This procedure is the most curative measure at our disposal. Though introduced in 1856 by Von Graefe, and practiced ever since that time, we do not yet know exactly how it produces the amelioration which follows in so many cases.

The operation of sclerotomy is favored by some. The incision of anterior sclerotomy is made just in front of the iris. In posterior sclerotomy it is between the ciliary body and equator. Removal of the superior cervical ganglion was introduced by Jonnesco. This operation has checked the progress of the disease in a large number of reported cases, and is becoming recognized as a very valuable procedure. If any constitutional condition exists which may be a factor in the production of the glaucoma it should receive appropriate

treatment. The local medicinal treatment is practically limited to the use of the miotics, eserin and pilocarpin. The sulfate or salicylate of eserin is used in strength varying from ¼ to 2 grains to ℥i. Pilocarpin may be used twice as strong. The frequency of instillation is determined by their effect on the pupil. For the pain use hot fomentations and anodynes.

Dionin, according to Darier, acts as a decided analgesic in glaucoma. He uses it in the same solution with the miotics. He also claims that the tension is favorably influenced both by the use of adrenalin and by gentle massage of the ball through the closed lid.

DIAGNOSTIC TABLE.

	CONJUNCTIVITIS.	INTERSTITIAL KERATITIS.	IRITIS.	ACUTE GLAUCOMA.
Redness.	Palpebral and ocular conjunctiva injected. Pericorneal zone the last part to get red.	Pericorneal zone injected. In bad cases the entire ocular conjunctiva becomes red.	Pericorneal zone injected first, entire ocular conjunctiva liable to become congested.	Diffuse redness with conjunctival chemosis and edema of the lid.
Pain.	Burning and scratchy as if there was a foreign body in eye.	Generally slight, but in some cases severe.	Sometimes absent but usually severe, following the fifth nerve into the cheek and forehead.	Always severe, following the fifth nerve into the cheek and forehead.
Vision.	Unimpaired or slightly diminished by mucus on the cornea.	Greatly diminished and same in all parts of the field of vision.	Diminished by turbidity of the aqueous humor but not as bad as in interstitial keratitis or acute glaucoma.	Greatly diminished; nasal side of field first and most.
Discharge.	Muco-purulent or purulent.	Watery.	Watery and profuse.	Watery.

Pupil.	Normal.	Normal.	Contracted and sluggish.	Dilated and of a green color.
Tenderness on pressure.	None.	None.	Slight. Considerable over the ciliary body if it be involved.	Great.
Tension.	Normal.	Normal.	Possibly slightly increased.	Greatly increased.
Temperature, pulse, etc.	Normal.	Normal.	Normal.	Increase in temperature and pulse, sometimes vomiting.
Cornea.	Normal.	Hazy, pink or buff colored sometimes hypersensitive.	Normal.	Hazy, with sub-normal sensitiveness.
Age of patient.	Any.	Disease of childhood rarely seen after 25 years.	Any age, but rare before puberty.	Rarely seen in patients under 35.

LESSON XXIV.

DISEASES OF THE CRYSTALLINE LENS.

DISLOCATION OF THE LENS.

This condition may exist congenitally or may be due to accident or disease. The lens may be partially held by the suspensory ligament or may be totally detached. The dislocation may be to the side, back into the vitreous or forward, through the pupil, into the anterior chamber. If due to disease it is associated with choroiditis, cyclitis and a fluid state of the vitreous. The lens may be perfectly transparent or

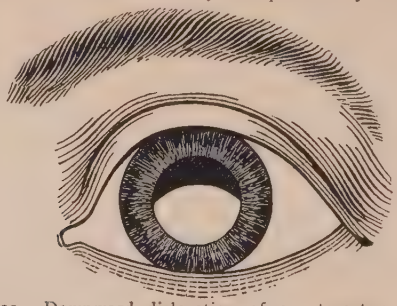

FIG. 119.—Downward dislocation of a cataractous lens.

cataractous. If cataractous the malposition may be easily detected (Fig. 119). If transparent and not in the anterior chamber the ophthalmoscope will reveal the condition. A transparent lens in the anterior chamber can be diagnosed by close inspection with the unaided eye. Congenital partial dislocation may be left alone. When due to accident or disease it would better be removed.

CATARACT.

Opacity of the lens, or its capsule, or both, constitutes cataract. Numerous terms, which explain themselves, are used

in classifying cataracts, such as lenticular, capsular and capsulo-lenticular; partial and complete; traumatic and spontaneous; fluid, soft and hard; congenital, juvenile and senile; immature, mature and hypermature; simple and complicated; stationary and progressive; gray, white, amber and black.

Cataracts are further classified by terms which indicate the location of the opacity as follows:—

1. **Anterior polar cataract,** in which the opacity is a small spot in the center of the anterior capsule, extending into the subjacent lens substance. It is usually pyramidal

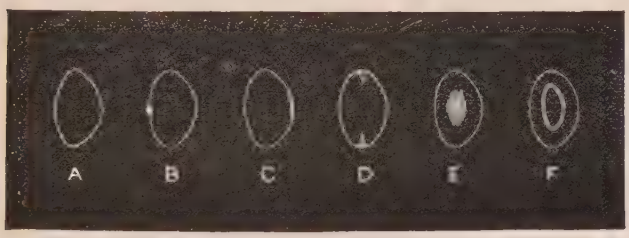

FIG. 120.—A, normal lens; B, anterior polar cataract; C, posterior polar cataract; D, cortical cataract; E, nuclear cataract; F, Lamellar cataract. (Juler.)

in shape, and may be congenital or acquired in early infant life. When congenital it is supposed to be the result of a fetal inflammation in which a deposit of lymph was left on the anterior capsule; or to be due to the adherence of the fetal pupillary membrane to the capsule. When acquired it is due to a perforation of the cornea which has allowed the lens to come forward in contact with the cornea.

2. **Posterior polar cataract,** similar to the preceding, except located in the center of the posterior capsule. In fetal life the hyaloid artery comes in contact with the lens at the posterior pole. An incomplete clearing up of the point of contact would explain a congenital posterior polar cataract. Another form of opacity generally called posterior polar but

which in reality lies in the posterior cortex assumes the shape of a star or rosette; the center of the star corresponding to the posterior pole of the lens. It is usually associated with retinal or choroidal disease.

3. **Lamellar or zonular cataract,** in which the opacity is confined to one or more of the layers of the lens. It is assumed that there is a disturbance of nutrition at a period of fetal life subsequent to the development of the clear nucleus. The layer of lens substance developed at the time of the nutritive disturbance is opaque. The interruption to the normal development of lens matter being temporary the subsequent layers are transparent.

FIG. 121.—Nuclear cataract. 1, Section of lens; central position of opacity; 2, Appearance by ophthalmoscope; 3, Appearance by oblique illumination.

4. **Nuclear cataract** in which the opacity begins in the hard center of the lens.

5. **Cortical cataract,** in which the opacity begins at the periphery of the lens.

Symptoms.—In children, if the cataract is complete, it is easily diagnosed, as the pupillary area will be white or gray and the eye will be blind. If the cataract is zonular, which is the most frequent form in children, and is always congenital or arises in early infant life, the vision is much reduced, the child behaving as if near-sighted. Best vision is secured when the pupil is dilated, as the patient can then see around the opacity. Hence these children will shade their eyes or turn their backs to the light to get better vision, and will find their sight improved by the twilight or cloudy weather. Close inspection, with the pupil dilated, will reveal a pale, round, central opacity of the lens; the rim of the

opacity being denser than the center, that will distinguish it from a nuclear cataract which is denser in the center. It is often found that children with zonular cataracts have been subject to infantile convulsions or are affected with rachitis. Anterior polar cataract is easily detected by the small, snow-white speck seen in the pupillary area. It does not affect vision as much as the zonular variety. The great majority of cataracts arise after the 45th year and are called senile or hard. The latter term is derived from the condition of the nucleous, which at this age has become quite dense. They are usually nuclear or cortical. As a rule the first symptom noticed is failing vision, not improved by glasses. In some

FIG. 122.—Cortical cataract. 1, Section of lens; opacities beneath the capsule; 2, Opacities as seen by the ophthalmoscope; 3, Opacities as seen by oblique illumination.

cases, in the first stages of the disease, the opacity increases the index of refraction of the lens, and thus increases its refractive power sufficiently to enable the patient to read without glasses. The patient rejoices in what is commonly called *second sight.* Unfortunately this state is temporary and gradual loss of vision follows. If the cataract is nuclear vision is improved by any circumstance which dilates the pupil. There is generally some hyperemia of the conjunctiva, lacrymation and itching. Sometimes inspection of the pupil reveals no anomaly, but cataract, in the advanced stage, shows a distinct white or grayish appearance of the pupillary area. Oblique focal illumination will demonstrate some opacities, but to determine their presence in doubtful cases the ophthalmoscope must be used. With the pupil dilated the whole lens can be brought under inspection and the slightest opacity will

be detected by the ophthalmoscope. The time between incipiency and maturity varies greatly in different cases, and in a few a partial opacity will remain stationary for the rest of life. A traumatic cataract, due to rupture of the capsule, may swell so rapidly from imbibition of aqueous humor as to bring on glaucomatous symptoms. A cataract is said to be ripe when all the lens substance is opaque. This may be determined by throwing a light, obliquely, into the dilated pupil. If there is a dark, crescent-shaped shadow on the lens, next to the iris, on the same side as the light, there is considerable lens substance which is still transparent. If the entire lens is opaque, no shadow will be cast upon it by the margin of the iris.

Cause.—Cataract is supposed to be due to some disturbance of the nutrition of the lens. It is a degenerative change coming as do gray hairs, very little being known of the conditions that conduce to it. Diabetes, ergotism, heredity, glaucoma, hyperopia, and spasms in children are supposed to be etiological factors. Glass blowers, stokers, etc., whose eyes are exposed to excessive heat and light are supposed to be unusually subject to the disease. Traumatic cataracts are due to some accident which punctures the capsule or loosens the lens from its ligamentous attachment.

Treatment.—Spontaneous absorption of the cataract has been reported in a few authenticated cases, but no therapeutic agent has yet been discovered that will bring about this happy result. The treatment is surgical. For the operations suited to the different varieties consult a text book. Before operating or recommending a case for operation be sure to test the bad eye for other pathologic conditions. Cataract prevents the distinction of objects, but does not obstruct light. Take the patient into a darkened room and with the good eye well covered see if he can point out the direction of a lighted candle when held in all parts of the

field of vision. If he can readily follow the light his retina and optic nerve are healthy and a successful operation will make him see again. If he can not see the light at all an operation is useless. If he sees only in certain parts of the field, or detects slowly the difference between light and shadow, an operation will be proportionately meager of results.

An *aphakic* eye is one which has no lens. The removal

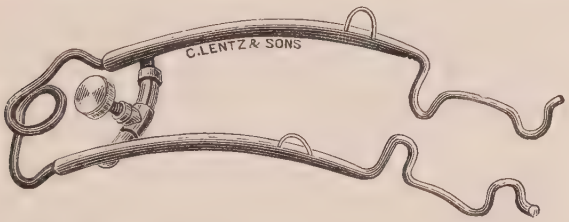

FIG. 123.—Speculum used to hold the lids open in operations upon the eyeball.

of the crystalline lens from an emmetropic eye will make it very hyperopic. After a cataract operation upon an emmetropic eye a strong convex lens must be worn to compensate for the one removed. If the operated eye was previously hyperopic the correcting lens will be stronger still; if it was myopic the correcting lens will be weaker than in the case of an emmetropic eye. It is possible for the removal of the lens to so neutralize a high degree of myopia as to secure excellent vision without the help of glasses (page 50).

LESSON XXV.

DISEASES OF THE RETINA.

HYPEREMIA AND ANEMIA.

The retinal vessels do not participate much in the changes of the intra-cranial circulation. There is some retinal congestion in meningitis and always venous engorgement in papillitis and thrombosis of a retinal vein. The same condition of the veins, in milder form, is often met with in emphysema and in weakness of the heart's action. Slight hyperemia of the retina and disc are sometimes associated with the strain of an uncorrected refractive error, but these mild hyperemias are difficult of diagnosis owing to the variations in the appearance of the fundus, found in health.

Anemia of the retina may result from embolism of the central artery, great loss of blood, cholera, spasm of the arterial coats due to toxic doses of quinin and from spasm due to vaso-motor disturbance. The dimness of vision found in some cases of migraine or "blind headaches" are examples of the latter condition.

RETINAL CHANGE FROM DIRECT SUN RAYS; SNOW BLINDNESS; ELECTRIC OPHTHALMIA.

Persons who have looked directly at the sun have sometimes complained afterward of a central scotoma. These blind spots vary in their severity and persistency, sometimes being permanent. There may be central defect for colors, and also metamorphopsia. The ophthalmoscope will often show a minute lesion near the macula. The treatment consists of rest of eyes, dark glasses and hypodermatic injections of strychnin.

As a rule the only result, if any, of exposing the eyes to the glare of the snow is a mild form of conjunctivitis, but sometimes there is temporary, and, in rare instances, permanent amblyopia.

Exposure of the eyes to strong electric light, as in electric welding, may result in severe changes, such as are found in injury by direct sunlight, and may take a mild form of ophthalmia, such as is occasioned by exposure to snow.

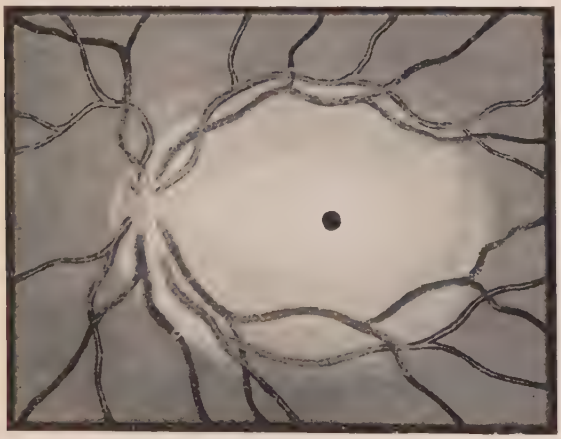

FIG. 124.—Embolism of the central artery of the retina. (Jennings.)

Electric workers now prevent these conditions by using glasses deeply colored with yellow, ruby or a combination of deep blue and red.

EMBOLISM AND THROMBOSIS OF RETINAL VESSELS.

Symptoms of Embolism.—Sudden loss of vision, partial if the obstruction lodges in a branch artery, total if it stops in the main trunk. The blood vessels will be much reduced in size. The retina will be white and opaque, the greatest opacity lying in the region around the macula and disc. The macula will appear as a cherry red spot, owing to the

fact that it is much thinner than the rest of the retina, and the choroid shows through it. Degeneration of the retina occurs in a few days, soon followed by atrophy. The optic nerve generally atrophies. Vision is rarely restored.

Cause.—The plug may come from vegetations in the heart, due to valvular disease or endocarditis. It may also

FIG. 125.—Thrombosis of the retinal veins. (Jennings.)

result from aneurism of the aorta or from atheroma of the arteries. It also occurs with Bright's disease and pregnancy.

Symptoms of Thrombosis.—The extent of visual loss depends upon the location of the thrombus in the central vein or one of its branches. Vision is, as a rule, not lost as suddenly as in embolism. There will be edema of the disc, tortuosity and engorgement of the veins, and numerous hemorrhages in the area drained by the thrombotic vein.

Cause.—Retarded venous circulation of the old, the emphysematous or those suffering with cardiac lesions. It may also be due to phlebitis.

Treatment of embolism and thrombosis is of little avail. An attempt may be made to absorb the obstruction by using iodid of potassium.

RETINITIS.

Inflammation of the retina may be limited to this membrane or may be associated with inflammation of the optic nerve (neuro-retinitis) or choroid (choroido-retinitis).

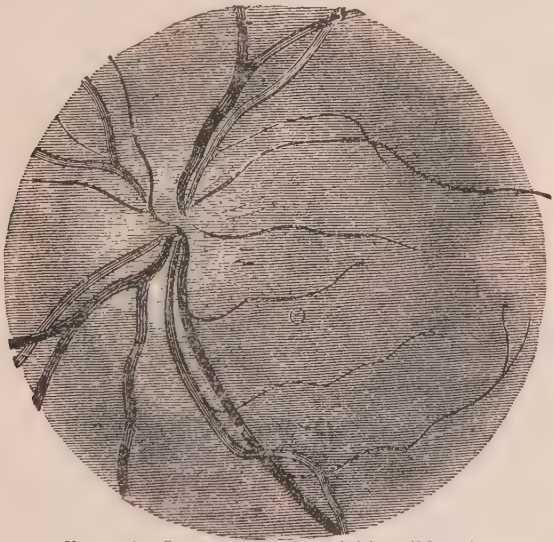

FIG. 126.—Serous or simple retinitis. (Meyer).

The disc is usually involved unless the retinitis is very mild, and some opacity of the vitreous often co-exists. Owing to the fact that the disease is generally constitutional in origin, we find it almost always bilateral. Normally the retina is a transparent membrane, but when inflamed it appears smoky or hazy and, at times, to such an extent as to obscure its vessels in some part of their course. The veins may appear unusually large and tortuous and frequently there are hemorrhages. Often there will appear distinct white spots

arranged along the course of the vessels or around the macula or disc. These spots can be differentiated from choroidal atrophy by the absence of the pigmented border and the softness of their outline. In retinitis there will be dimness of vision in all degrees. There may be limitation of the field of vision and perhaps scotomata. Micropsia (objects appearing unnaturally small), megalopsia (objects unnaturally large), metamorphopsia (unnatural posi-

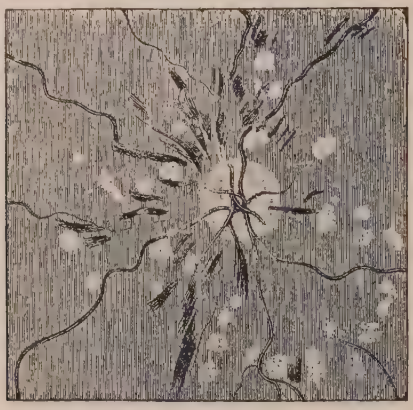

FIG. 127.—Recent severe retinitis in renal disease. (Gowers.)

tion of objects in the field, straight lines appearing wavy, etc.) and night blindness are forms of visual disturbance which may be manifested. There may be photophobia but there will be no pain and no external evidence of inflammation. Recovery may take place with little or no loss of vision but generally the prognosis is grave. The result depends largely upon the cause of the attack, and the region of the retina involved. Different forms of the disease are named according to etiology, leukemic, albuminuric, diabetic, gouty and syphilitic. There are other forms named from characteristic features such as simple, hemorrhagic and purulent retinitis.

Albuminuric retinitis occurs in about seven per cent. of all forms of albuminuria. It appears late in the stage of the renal trouble, the majority of patients dying within two years after its advent. Both eyes are generally affected. The characteristic feature is the presence of the white spots of fatty degeneration which tend to arrange themselves in a stellate figure around the macula. Complete recovery from albuminuric retinitis has been observed.

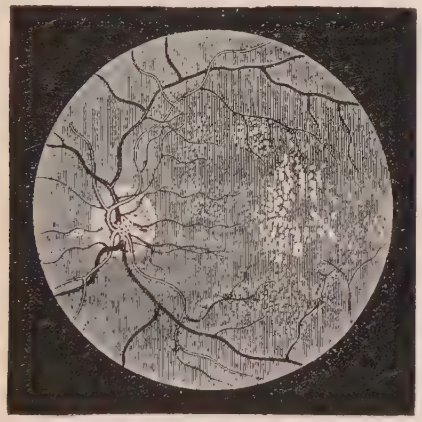

FIG. 128.—Renal retinitis at a late stage. (Wecker and Jaeger.)

Diabetic retinitis presents the same general symptomatic picture as albuminuric retinitis. If there is any difference it is that the white degenerated spots are more diffuse and have not the same tendency to a stellate formation around the macula in diabetic retinitis. Hemorrhages are also more numerous in the latter form. Often the differentiation will depend upon the urinary analysis.

Syphilitic retinitis occurs from congenital as well as acquired syphilis. It is, as a rule, associated with choroiditis and opacity of the vitreous. Night blindness is a prom-

inent symptom. The general characteristics are those already described without the white spots of fatty degeneration, which are almost pathognomonic of renal disease. See syphilitic choroido-retinitis, page 145.

Hemorrhagic retinitis, as the name implies, is an inflammation of that tissue in which hemorrhages are the most prominent feature. These flame-like extravasations may be scattered over the whole fundus. The optic disc is usually edematous, the veins tortuous and enlarged. Vision depends largely upon the location of the hemorrhagic spots in relation to the macula. The disease appears after middle life and in those suffering from disease of the heart or of the vessel walls. The ophthalmoscopic picture is practically identical with that of thrombosis of the retinal vein and differentiation may be impossible.

Cause.—Generally one of the constitutional conditions enumerated above is responsible for the disease. The etiology of simple, idiopathic retinitis is obscure.

Treatment.—Demand absolute rest of the eyes. Subdue the light by the use of smoked glasses. Treat the constitution as indicated by each case.

RETINITIS PIGMENTOSA.

This is a degenerative rather than an inflammatory condition. It is extremely chronic in its course, sometimes requiring years to reach its usual termination in blindness. Vision is much affected, but the symptom most complained of is night blindness. The field of vision gradually contracts until only central vision is left. This much may remain for years. The fundus shows a peculiar stellate pigmentation beginning at its periphery and extending gradually to the macula. The amount of pigment is no measure of the gravity of the case. The calibre of the retinal vessels diminishes and there is slow atrophy of the

retina and disc, with occasional opacity of the posterior lens capsule.

The cause is obscure, but consanguinity of parents seems to be an etiological factor. No treatment is successful. Galvanism and strychnin have been recommended.

FIG. 129.—Pigmentary degeneration of the retina. (Jaeger.)

DETACHMENT OF THE RETINA.

This condition consists in a separation of the retina from the choroid, the intervening space being occupied by a serous fluid, blood or a tumor. Vision is affected in proportion to the extent and location of the detachment, the field showing a defect corresponding to the position of the lesion. The ophthalmoscope reveals a steel gray reflex from the detached part, over which the retinal vessels flow. The presence of the vessels distinguishes this from any other condition presenting a similar reflex. If the detachment be recent there will be partial loss of vision, which increases with the degeneration of the retina. The retina rarely returns to its normal condition. The size of the detachment

may remain stationary and may extend over the entire fundus.

Cause.—Blows upon the ball or jars by transmitted force. Tumors of the choroid. Disease resulting in fluidity or shrinkage of the vitreous. High degrees of myopia.

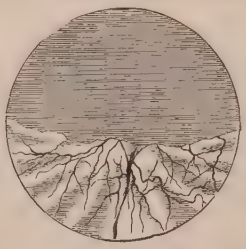

FIG. 130.—Ophthalmoscopic appearance of detached retina (erect image). After Wecker and Jaeger.

Treatment.—Long continued rest in bed with eye bandaged. Evacuation of the subretinal fluid by a knife, needle or pointed cautery; or absorption of it by pilocarpine sweats and abstinence from fluids. The results of treatment are discouraging.

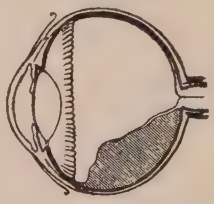

FIG. 131.—Section of eye with partial detachment of retina. (Nettleship.)

GLIOMA OF THE RETINA.

This very malignant tumor is either congenital or appears in infancy. The first symptom noticed is a yellow reflex from the pupil. Tests will prove the eye to be blind. It is difficult to differentiate from pseudo-glioma in this stage (page 147). Owing to the great rapidity of the tumor's growth the diagnosis can soon be made. Active inflamma-

tion, increased tension and pain soon develop with glioma; whereas in pseudo-glioma there is no pain and the tendency is toward diminished tension and shrinkage of the ball. In a few months the tumor fills the eyeball and with its continued growth protrudes between the lids, fills the orbit and

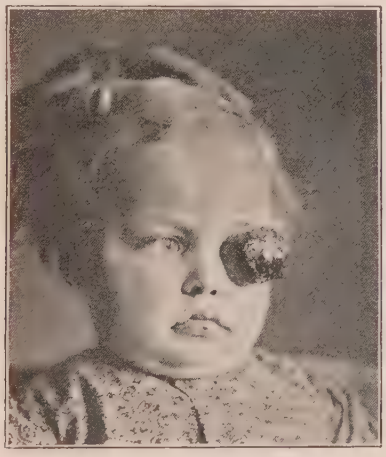

FIG. 132.—Glioma of the retina.

extends to the brain. Distant organs may become affected. If the eyeball is removed when the tumor is confined within it, there is some hope for the patient. When in doubt as to the diagnosis, enucleate as no harm is done if the microscope proves the case to be pseudo-glioma.

LESSON XXVI.

DISEASES OF THE OPTIC NERVE.

Optic neuritis, or inflammation of the optic nerve, may be divided into two kinds:—

1. Papillitis, which involves the intra-ocular end of the nerve.

2. Retro-bulbar neuritis, which affects the nerve between the ball and the chiasm.

PAPILLITIS.

The optic disc or papilla is the intra-ocular termination of the nerve or that part between the lamina cribrosa and the retina. With the ophthalmoscope the disc appears as a white, circular area in the orange colored groundwork of the choroid. The white reflex of the disc is caused by the lamina cribrosa showing through the transparent nerve fibers. In inflammation of the disc there are no definite subjective signs. There is usually contraction of the field of vision and derangement of color perception, but vision may not be reduced until late in the progress of the disease. Main reliance in diagnosis is placed on the ophthalmoscope, which shows a serous infiltration of the disc manifested by redness, swelling and loss of its outline. The retinal arteries appear small and the veins filled and tortuous. The strangulation of the veins sometimes results in hemorrhages in the retina. In the great majority of cases both nerves are inflamed. If the inflammation extend by continuity of tissue to the retina, the condition is called *neuro-retinitis.* Papillitis may sometimes result in complete recovery, but in the majority of cases a partial or total atrophy of the nerve results.

Cause.—If monolateral it is generally the result of some orbital lesion. When bilateral it is usually due to intracranial disease, most frequently to tumors, but it may be due to meningitis, abscess, depressed fracture or softening. It may also be the result of albuminuria, diabetes, syphilis, lead poison and anemia. The prognosis will depend largely upon the etiology.

FIG. 133.—Ophthalmoscopic view of the disc in optic neuritis. The outline of the disc is clouded. The retinal arteries are contracted, the retinal veins are dilated and tortuous and both are hazy in places. (Fuchs.)

Treatment.—Forbid use of eyes. Direct your efforts to the cause, and when in doubt give iodid of potassium, and build up the constitution by the usual methods.

RETRO-BULBAR NEURITIS.

This condition is also called central amblyopia and toxic amblyopia.

Symptoms.—Loss of acute vision, the patient complaining of a mist before the eyes. There will be central scotoma for red and green, and in advanced cases, central scotoma for objects. The affection is nearly always binocular and the vision of the two eyes nearly the same. There is

no contraction of the field, which aids in the differentiation from progressive atrophy. In pronounced cases, the disc shows an unnatural whiteness of its temporal side, and in an advanced state the whole disc may present the appearance of atrophy. There is proliferation of connective tissue in the nerve and atrophy of those fibers which go to the macula. The progress of the disease is slow, and the chance of recovery good unless of too long standing. It is almost exclusively a disease of men.

Cause.—It is due, in the great majority of cases, to the excessive use of tobacco or alcohol. Many observers con-

FIG. 134.—Choked disc, longitudinal section. (Fuchs.)

sider tobacco the most potent etiological factor, and some entirely acquit alcohol of any responsibility for the disease. Alcoholic extract of Jamaica ginger, essence of peppermint, etc., are very popular in some prohibition regions and a number of serious cases of amblyopia have been reported from their use. The methyl alcohol which they contain is considered the toxic agent. Exposure to cold, diabetes, rheumatism, syphilis and poison by some chemicals, among which are iodoform and bisulfid of carbon, are supposed to be causative; and there are also some cases in which no cause can be discovered.

Treatment.—Absolute abstinence from the offending poison. Watch the patient's digestion and give iodid of potassium or strychnin.

QUININ AMBLYOPIA.

Overdoses of quinin cause another form of toxic amblyopia in which the symptoms are so different from those just enumerated, as to merit a separate description.

Symptoms.—The general symptoms of cinchonism will precede the loss of vision. The amount of visual disturbance varies from a blurring of objects to absence of light perception. The pupils may be widely dilated and the ophthalmoscope reveal a picture similar to that presented by

FIG. 135.—Optic neuritis with hydrops of the optic nerve sheaths. (After Pagenstecher and Genth.)

embolism of the central artery. When there is sufficient vision to make a test possible, the field will be found contracted and color perception lost. After a variable period vision is decidedly improved though it rarely returns entirely to the normal. Salicylate of sodium and acetanilid can also produce a similar amblyopia.

Cause.—The action of the drug on the vaso-motor centers is supposed to be such as to cause a violent constriction of the blood vessels. An anemia of the retina is the result.

Treatment.—Use of the offending drug should be discontinued at once. Inhalations of nitrite of amyl may be tried. Strychnin should be given until constitutional effect is felt. Absolute rest of the eyes must be enforced and general health promoted in every way.

ATROPHY OF THE OPTIC NERVE.

In atrophy the medullary part of the fibers is displaced by granular fat and connective tissue, with thickening of the walls of the vessels and narrowing of their calibre.

Symptoms.—There is no pain and no change in the appearance of the ball, unless total blindness exists, when the pupils will be dilated. Great variety is manifested in the visual defects. The loss of vision may be rapid but is generally very gradual and slow. Central vision is lowered

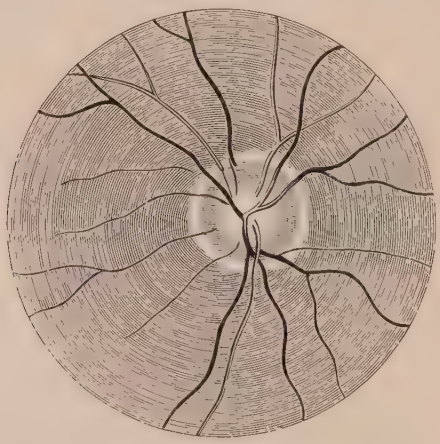

FIG. 136.—Atrophic excavation of the disc.

and the field contracts, sometimes concentrically, sometimes irregularly. Color sense becomes defective or lost, perception of green, red and blue usually disappearing in the order named. If the atrophy be associated with spinal cord lesions we shall, as a rule, find the Argyll-Robertson pupil. The disc will appear abnormally white or gray, slight excavation due to shrinkage of its substance may be noticed and the retinal vessels will be reduced in size.

Cause.—Spinal diseases of which tabes dorsalis is the most important, papillitis, pressure of tumors, disease of the

orbit, thrombosis and embolism of the retinal vessels, glaucoma, meningitis, syphilis, alcoholism and anemia from great loss of blood may cause atrophy of the optic nerve. It may appear as a purely local disease independent of any other lesion.

Treatment.—Correct any derangement of general function. The galvanic current, one pole over the eye and the other at the back of the neck, is of doubtful utility. Give iodid of potassium, mercury or strychnin to the point of tolerance. Strychnin is more efficacious when given hypodermatically. Antipyrin, seven and a half grains every other day, hypodermatically is also recommended. Treatment is usually ineffectual.

LESSON XXVII.

FUNCTIONAL DISORDERS OF VISION, ETC.

AMBLYOPIA AND AMAUROSIS.

These are terms used to express a diminution or loss of vision without any apparent lesion. The two words are used, more or less indiscriminately, but amaurosis is generally applied to the graver conditions. There are many forms of lowered vision, the pathology of which is known, but they do not come under this head. The cause of functional amblyopia may be known but the exact way in which the loss of vision is produced is unexplained.

1. **Traumatism** to the head, direct or indirect, or a blow upon the eye, may be followed by amblyopia, more or less persistent. In these cases it is presumed that there is some invisible lesion of the parts concerned in vision.

2. **Loss of blood** may produce amblyopia, which probably is due to the fact that the retina is affected by the general lack of nutrition. But we do not know why the degree of amblyopia is not always commensurate with the amount of blood lost or why there is greater tendency to amblyopia from hemorrhages of the stomach, uterus and bowels than from traumatic hemorrhages.

3. **Congenital amblyopia** generally affects but one eye. It has been mentioned as a factor in the production of concomitant strabismus. It is supposed to be due to an arrest of the development of the eye in fetal or early infant life. Lowered visual acuity is often associated with pronounced errors of refraction, especially astigmatism. If accurately corrected at an early age the eye may gradually develop normal acuteness of vision.

4. **Hysterical amblyopia** may occur in both sexes but is most frequent in females. As might be supposed the symptoms assume a great variety of forms, such as total blindness, hemianopsia, scotoma, color blindness and contraction of the visual field. Neurasthenic school children, especially girls, are frequently thus afflicted and great tact and judgment are required in their treatment.

5. **Simulated amblyopia** or malingering, may be due to a desire to exaggerate an injury over which a lawsuit is pending, to secure a pension, to escape some disagreeable duty or to excite sympathy. For obvious reasons, but one eye is claimed to be affected. Numerous tests will reveal the patient's hypocrisy, if he claims blindness in but one eye, among which are the following:—

1. Put on him a pair of spectacles, one lens of which is plain glass and the other a prism with its base up or down. If malingering he will see double and an effort to walk, especially to go down stairs, will be made so cautiously that his true condition is detected.

2. Place before the eye he claims is bad a plain glass and before the other a plus glass just strong enough to obscure its vision. If with these, vision is normal, the patient is malingering.

3. Hang some green letters in front of a *black* background, at a convenient distance. Hold before the good eye a glass colored red. If he reads the letters, he does it with his bad eye, as the green letters cannot be seen through the red glass. Red letters on a *white* background are much obscured when seen through a red glass.

4. Put a drop or two of atropin in the good eye and to allay suspicion an equal number of drops of cocain may be put in the bad eye. When time enough has elapsed for the atropin to paralyze accommodation, hand the patient a book.

If he reads it he does so with the eye he claims to be amblyopic.

If the patient claims to be blind in both eyes his detection is more difficult and a close watch may be necessary to determine the true condition. A simple test, which may be of service, is to ask the patient to look at his own hand. A blind man will turn the eyeballs toward the hand, a malingerer may intentionally look in some other direction.

AMAUROSIS PARTIALIS FUGAX.

This condition, which is also known as scintillating scotoma is a very common form of temporary disturbance of vision. It may not be recognized owing to the unsatisfactory description of his symptoms given by the patient. A sudden blurring of the vision is noticed, which for a period of five minutes gradually gets worse. Reading is then almost impossible and distant objects have a veiled and confused outline. There is a very uncomfortable flickering or glimmering appearance in the field of vision. The sensation is likened to that presented by the atmosphere which rises over a red hot stove. There may be flashes like zigzag lightning circling around the periphery of the field, and homonymous hemianopsia may be present. The uncomfortable sensation is not dispelled by closing the eyes. In about thirty minutes the symptoms disappear and are followed in the majority of cases by a frontal headache. The attacks vary in frequency from one in a life time to several a day. It is supposed to be due to a circulatory disturbance in the brain cortex. Patients will attribute it to an indefinite variety of causes. Treatment consists in improving the general condition of the patient. Validol in 20-drop doses has been recommended.

HEMIANOPSIA.

Hemianopsia is a term used to express diminution or total loss of vision in one-half of the field. Both eyes are usually

affected, which indicates a lesion in or back of the chiasm. If only one eye is affected the lesion is probably in front of the chiasm. In the great majority of cases of hemianopsia affecting both eyes the diminution or loss of vision will be in the right half of each field or the left half of each field. This is called *homonymous* hemianopsia. We may have loss of the external half of each field called *bitemporal* hemianopsia, or of the internal half of each field called *binasal*

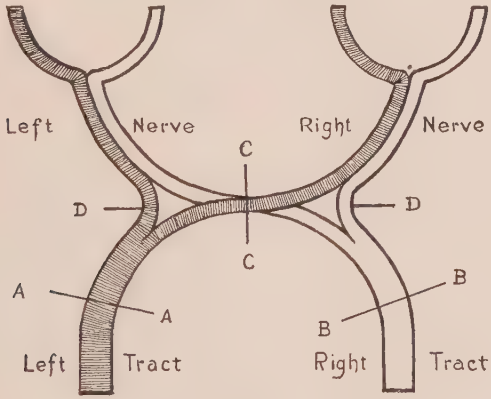

FIG. 137.—Lesion at A, A, would cause right homonymous hemianopsia. B, B, would cause left homonyous hemianopsia. C, C, would cause bitemporal hemianopsia. D, D, would cause binasal hemianopsia.

hemianopsia, but these conditions are rare. A condition still more uncommon is loss of the upper or lower field. The lesion which causes hemianopsia may lie at any part of the visual tract from the eyeball to the cortex of the brain and a knowledge of the origin and distribution of the optic nerve fibers is necessary to determine its location. The lesion may be a tumor, periostitis, blood clot, softening of the brain, atheroma of adjacent vessels, injuries, etc. Treatment must be directed to the cause.

NIGHT BLINDNESS.

Most authors use the word hemeralopia to express night blindness and nyctalopia to express day blindness. Green-

hill has proven that they are in error and that the reverse is correct according to derivation and ancient usage. The night blindness of retinitis pigmentosa must not be confounded with the *functional* variety being described in which there are no visible lesions of the fundus. In functional night blindness vision may be normal in a bright light but is greatly lowered on dull days, in the twilight or in dimly lighted rooms. It is found in persons who have been exposed to glaring light, such as travelers in the tropics, glass blowers, electric welders and those who work before fur-

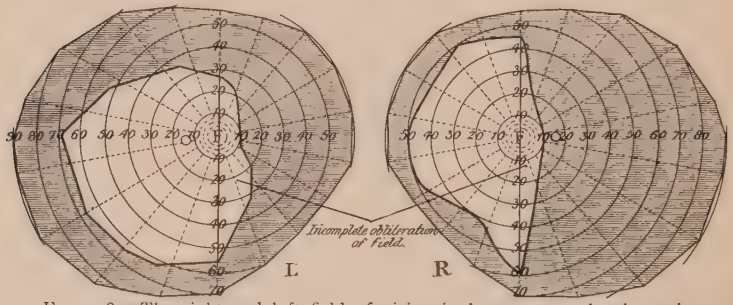

FIG. 138.—The right and left field of vision in homonymous hemianopsia, resulting from unilateral cerebral hemorrhage. The darkened areas indicate the obliterations. The normal areas remaining are contracted.

naces. It is also supposed to be associated with certain states of lowered vitality such as scorbutus, starvation, etc.

The treatment consists in protection of the eyes by dark glasses, the use of tonics of quinin, iron, strychnin and cod liver oil, and changing the occupation if that seems at fault.

DAY BLINDNESS.

This condition is the opposite to night blindness in that the patient sees better and greatly prefers diminished illumination. It is found as a symptom in retro-bulbar neuritis, albinism, dilatation of the pupil from third nerve paralysis or from the use of a mydriatic, central cataract, etc. It also exists as a *functional* condition independent of any demon-

strable lesion. Functional day blindness is found in persons who have been excluded from the light for a long period and is also a frequent symptom of hysteria.

When day blindness is a symptom, the primary affection should be treated. If it is functional examine closely for muscular insufficiency and errors of refraction; build up the constitution and accustom the eyes to light by resorting to the strategy demanded by each case.

LESSON XXVIII.

GENERAL THERAPEUTICS.

THE APPLICATION OF HEAT AND COLD.

Heat is of great service in relieving pain in and around the eye. Dry heat is best applied by means of a Japanese box. This is a small tin case which can be kept hot for several hours by means of a burning cartridge of punk. Flannel cloths, heated before a fire or in an oven, are efficacious, but require frequent changing.

Moist heat is secured by cloths wrung out of very hot water. They should be changed often enough to maintain an equable temperature. Another way of applying moist heat is to fill a tumbler to the brim with hot water and then place the eye, with the lids closed, as far in it as possible. Moist heat by stimulating the circulation promotes resolution, as, for example, in ulceration of the cornea. It hastens the suppurative process as in phlegmon of the lid. It is applicable to all deep-seated inflammations, such as iritis and glaucoma. Heat is usually applied for fifteen to thirty minutes at a time, three or four times a day.

Cold is of service in restraining the inflammatory process after traumatisms and in reducing the congestion of conjunctival inflammations. Muslin squares may be placed on a block of ice and transferred to the eye with sufficient frequency to maintain the desired temperature. The desired effect may also be secured by wringing the cloths out of cold water and changing them frequently. The cold application on page 94 is very grateful.

ANESTHETICS.

General anesthesia should be used on unruly patients, in plastic operations, enucleations, advancements of the mus-

cles and when there is so much inflammation that local anesthetics are ineffective.

Local anesthesia will suffice for the majority of eye operations. The removal of chalazions and other lid operations are not rendered painless by the instillation of a local anesthetic. It must be injected into the tissues and this is often impracticable owing to the change produced in the relationship of the parts.

Cocain hydrochlorate is used in two to four per cent. solutions. If greater strength is needed its toxic properties must not be forgotten. The indiscriminate use of cocain in the treatment of eye diseases is to be deplored. It is questionable if it serves a good purpose in any other role except that of an anesthetic. There is no doubt of its ability to do harm under certain conditions. It causes a drying out and exfoliation of the epithelial layer of the cornea. To prevent this the patient may keep the lids closed between instillations and in operations some bland aseptic solution may be dropped on the cornea at proper intervals. Cocain also reduces the intra-ocular tension, constricts the small blood vessels and dilates the pupil. For the removal of foreign bodies from the cornea, one drop is often all that is necessary. A second drop a minute later will quite suffice. For the formidable operations more instillations are required. Cocain spoils quickly and old solutions should not be used.

Holocain hydrochlorate is used in a one per cent. solution. It causes slight pain when first dropped in the eye. It does not reduce congestion or lessen hemorrhage by constricting the blood vessels as does cocain. Its advantages over cocain are that it does not dilate the pupil, causes no drying of the cornea, will not spoil in solution, and has decided antiseptic properties. Holocain should not be used hypodermatically. The bottle should be washed out with pure muriatic acid before a solution of holocain is put in

it, otherwise a combination with the trace of lead, always found in a new bottle, will take place.

Eucain "B" has proven too irritating to supplant either of the above anesthetics in eye practice.

Chloretone, Anesin and **Acoin** are new local anesthetics which possess no properties which specially recommend them.

MYDRIATICS.

Mydriatics are drugs which dilate the pupil.

Cocain hydrochlorate may be used when a temporary dilatation is required as, for example, for ophthalmoscopic examinations. The eye must be kept closed while the pupil is dilating and too much cocain must not be used or a wrinkling of the epithelial layer of the cornea will prevent the examination. Cocain does not cause paralysis of the ciliary muscle, consequently vision is very slightly affected by its use. The pupil will return to its normal state in an hour or two.

Euphthalmin hydrochlorate is very similar in its mydriatic action to cocain. It does not dry out the corneal epithelium or paralyze the ciliary muscle. Euphthalmin is used in a five per cent. solution.

Ephedrin hydrochlorate is recommended as a useful mydriatic which will dilate the pupil without causing paralysis of accommodation. It is used in a five per cent. solution.

MYDRIATICS AND CYCLOPLEGICS.

A cycloplegic is a drug which causes paralysis of the ciliary muscle and resulting suspension of accommodation. All of the cycloplegics are also mydriatics.

Homatropin hydrobromate is a satisfactory mydriatic. Its effect upon the pupil is more lasting than that of cocain or euphthalmin. For mydriasis one grain to the ounce is sufficient. When homatropin is used as a cycloplegic, in testing for refractive errors, the mistake is often made of

using it too weak. The following prescription, freshly made, will produce total cycloplegia.

℞

Homatropin hydrobromate . . . . . gr. I.

Aq. destill. . . . . 3 ss.

Sig.—One drop in each eye every ten minutes, for one hour.

Thirty minutes after the last drop the patient is ready to examine. The effect will wear off in about thirty-six hours.

Atropin sulfate is the most powerful mydriatic and cycloplegic. A one per cent. solution is usually employed. Its effect will not disappear for about ten days, therefore it must not be used thoughtlessly on those who need their near vision. It is used when total inactivity of the ciliary muscle or wide dilation of the pupil is desired. Atropin is the active principle of belladonna and caution must be observed in its use as some individuals manifest a pronounced susceptibility to belladonna poison.

Duboisin sulfate (gr. ¼ to 3i.), **hyoscyamin hydrobromate** (gr. ¼ to 3i.), **scopolamin hydrobromate** (gr. ⅛ to 3ii.), and **daturin sulfate** (gr. ⅛ to 3i.) are cycloplegics whose action and use are about the same as atropin. Their effect wears off in from four to seven days. The use of a mydriatic has brought on glaucoma in the manner described on page 155. For this reason tension of the eye should be noted and great caution observed before their use, particularly in elderly people.

MYOTICS.

Myotics contract the pupil and increase accommodation by stimulation of the ciliary muscle.

Eserin sulfate or **salicylate** is the most powerful of the two myotics and is used in strength varying from gr. 1/20

to gr. ii. to ʒi., depending upon the object to be attained. The weakest dose is used for the purpose of stimulating accommodation and the strongest in glaucoma, page 157. Eserin solutions become pink when kept for any length of time, this does not indicate any deterioration in strength.

Pilocarpin hydrochlorate is much weaker than eserin and is used only when feeble action is desired. Grain i. to ʒi. is the strength generally used. It is also used hypodermatically to produce diaphoresis, for example, in detachment of the retina and in choroiditis.

ANTISEPTICS AND DISINFECTANTS.

It has been demonstrated that the thorough mechanical cleansing of the conjunctival sac with sterile water or normal salt solution is as successful a method of asepticizing it as the use of the chemical antiseptics. Antiseptics can not be used freely in strong solutions owing to the danger of irritating the conjunctiva and injuring the cornea. The strength employed will depend upon the object to be attained. The same antiseptic may be used in a weak solution to remove secretion from the conjunctival sac, in a concentrated form to disinfect a corneal ulcer and in an intermediate strength as an application to the palpebral conjunctiva.

Formalin, 1 to 6,000 solution, may be used in the conjunctival sac. As a disinfectant, applied directly to a corneal ulcer, on a cotton applicator, 1 to 100 may be used.

Trikresol is one of the least irritating antiseptics. A solution of 1 to 1,000 makes a valuable menstruum for eye drops as bacteria will not develop in it.

Peroxid of Hydrogen is exceedingly painful. It is used to disinfect corneal ulcers, but holocain anesthesia should precede it.

Bichlorid of Mercury should not be stronger than 1 to 5,000 if employed as a wash. It sometimes irritates in this

strength. When applied to a trachomatous lid it may be used as strong as 1 to 500, but its action must be limited to the part treated.

Biniodid of Mercury is used as a wash in *Panas' solution*, as follows:

Mercuric iodid	gr. $^1/_7$
Alcohol	℥i.
Aq. destill. q. s.	℥vi.

Permanganate of potash solution is generally employed in 1 to 2,000 strength.

Pyoktanin (blue) in 1 to 2,000 solution is supposed to be an especially reliable antiseptic in purulent conditions of the lacrymal sac.

Boric acid gr. xii. to ℥i., is used as a cleansing wash and as a collyrium. In the latter case it is generally combined with other remedies. Its antiseptic properties are quite limited.

Borax, gr. iv. to ℥i., is similar in its application to boric acid.

Carbolic acid is never used as a cleansing wash or as a collyrium. It is a popular disinfectant for corneal ulcers. When thus applied a local anesthetic must be used and great care observed to prevent the acid from spreading beyond the edge of the ulcer. Dip a lacrymal probe in pure carbolic acid and let as much run off as will; what remains may be sufficient. It is better to repeat this process a number of times than to apply so much that it runs over the healthy cornea.

Tincture of iodin is used in infected corneal ulcers the same as carbolic acid.

Silver nitrate is an old and valuable disinfectant and astringent. The preparations of silver are probably the most useful remedies we have in the treatment of eye dis-

eases. They are used as prophylactics, also in conjunctival inflammations particularly when pus is present, in disease of the lid border, in diseases of the lacrymal sac, and in ulceration of the cornea. One per cent. solution of nitrate of silver is dropped into the eyes of infants to prevent ophthalmia neonatorum. In two per cent. strength it is applied to the palpebral conjunctiva. To disinfect ulcers of the cornea it is used in strong solution in the same manner as carbolic acid. The "mitigated stick" is composed of silver nitrate and potassium nitrate in various proportions.

Protargol, a new organic salt of silver, is used in five to twenty per cent. solutions. It is not as irritating as silver nitrate and seems to be more efficient in some conditions.

Argentamin, Albargin and **Nargol** are new silver preparations which have their advocates but there seems to be no reason why they should supplant protargol.

Argyrol (silver vitelline) is a justly popular new silver product. It is painless and non-irritating, contains thirty per cent. of silver, does not coagulate albumen and is said to have greater penetrating action than any of the other silver preparations. It is used in five to fifty per cent. solutions. The latest reports on the action of argyrol are most favorable.

The Actual Cautery is a most useful disinfectant of corneal ulcers.

STIMULANTS AND ASTRINGENTS.

Copper sulfate crystal ("blue stone") is applied to the palpebral conjunctiva in trachoma and chronic conjunctivitis. The membrane should be touched lightly or rubbed with the crystal, depending upon the effect desired. The surface of the crystal must be absolutely smooth to avoid scratching the conjunctiva. It is sometimes employed in solution of one-half grain to the ounce.

Cuprol is an organic combination of copper and nucleinic acid, containing about six per cent. of metallic copper. The ten per cent. solution is generally used in conjunctival diseases.

Alum is employed in chronic conjunctivitis in the crystal form or in a solution containing from one-half a grain to two grains to the ounce.

Tannin dissolved in glycerin in the proportion of one part of tannin to eight of glycerin is sometimes applied to the palpebral conjunctiva in trachoma. It is also recommended in phlyctenular conjunctivitis.

Zinc sulfate is used in collyria, in strength varying from one to two grains to the ounce. It is of special value in diplo-bacillus conjunctivitis.

Zinc chlorid, grain one to the ounce, is also very useful in this disease.

Boroglycerid (U. S. P.) is applied to the everted lid in trachoma and chronic conjunctivitis.

Camphor water, a weak stimulant and astringent, is used in collyria. One part of camphor water to three of distilled water makes a good menstruum for stronger astringents.

POWDERS.

Powders must be triturated as thoroughly as possible before being dusted upon the cornea or used in an ointment in the conjunctival sac.

Calomel is dusted upon the cornea in cases of corneal ulceration, pannus and phlyctenular ophthalmia. It should not be used when an eye is in a state of active inflammation or when the patient is taking iodides internally, see page 118.

Iodoform is used in corneal ulceration. It may be applied as a powder or in an ointment. It sometimes produces severe irritation if care is not observed in the selection of the preparation used.

Xeroform and **Nosophen** are antiseptic powders which are recommended in the treatment of corneal ulceration.

OINTMENTS.

Yellow oxid of mercury is employed in the form of an ointment made with vaselin, cold cream or lanolin. When it is to be used in the conjunctival sac it will mix better with the tears if albolene is incorporated. The ointment should be *rubbed* until not a grain of the mercury can be seen. One per cent. is the usual strength, but it may be made stronger or weaker. It is used in diseases of the lids, conjunctiva and cornea as described under these subjects. It should not be exposed to the light.

Ammoniated mercury is indicated in the same conditions and is employed in the same strength as the yellow oxid.

Boric acid, one grain to the dram of vaselin, is a bland ointment which may be used on the border of the lids to prevent them from sticking together when secretion is excessive.

MISCELLANEOUS REMEDIES.

Jequirity is used to establish an acute membranous conjunctivitis in trachoma and pannus. It seems to have a decidedly beneficial influence in selected cases. Twenty grains of the decorticated beans are ground, then put in an ounce of cold water and allowed to stand for twenty-four hours. The palpebral conjunctiva is painted with this infusion and in a few hours active inflammation sets in. In a few days the inflammation subsides and gradual improvement of the pannus follows.

Jequiritol, a preparation sold by Merck, with full instructions for its use, is said to be safer and more reliable than the infusion of jequirity beans.

Suprarenal extract will produce a thorough hemostasis of the conjunctiva. Tenotomies, pterygium operations, etc., are rendered practically bloodless by its use. *Adrenalin*

chlorid and *Suprarenalin* are preparations of the gland which may be used in strength varying from 1 : 5,000 to 1 : 20,000. The value of suprarenal extract as a therapeutic agent has not yet been established.

Dionin is a morphin derivative used generally in a two per cent. solution. It causes pain when instilled into the conjunctival sac and should be preceded by a local anesthetic. It is a lymphagogue and a few minutes after its application a decided conjunctival edema appears. It is of decided value in relieving the pain of iritis and iridocyclitis. It is also used for the same purpose in ulceration of the cornea and glaucoma. It is said to influence the absorption of the post-operative debris of cataract. In dionin we undoubtedly have a valuable agent. Its applicability and limitations have not yet been well defined.

Fluorescin is used to determine the area of the cornea which is denuded of its epithelium by ulceration or traumatism. A drop of the following solution placed upon the cornea will stain green all tissue not covered by epithelium.

Fluorescin	gr. i.
Sodii bicarb.	gr. ii.
Aq. destill.	ʒ ii.

Cassaripe is the juice of the cassava plant. It is used in the form of a ten per cent. ointment in cases of ulceration of the cornea.

Ichthyol, in a 25 to 50 per cent. solution, has been used in conjunctival and corneal affections. It has been recommended for marginal blepharitis, in a ten per cent. ointment **Ichthalbin** is proposed as a substitute for ichthyol as it is free from the disagreeable odor of the latter.

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